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AMEBIASIS, A CLINICAL EVALUATION*

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There is reason to feel that we are threatened with "Amebaphobia." Our newspapers stimulated this fear with an alarming "approximation" of the prevalence of this parasitization locally. Many, failing to concur in this belief, have after adequate surveillance of our own practice come to a point of antagonistic denial of an untruth. This presentation is therefore an evaluation in abstract of clinical amebiasis as it presents itself in our valuation of recent publications, in a review of our own cases and in a comparative evaluation with others locally.

"The habitation of tropical outposts by our military personnel, imprisonment of Axis prisoners captured from these sections in our country, and the eventual return of our forces may produce an unwelcome familiarity with tropical disease throughout the nation."[1] This statement was the premise and essentially the conclusion of our presentation "Statistical Evaluation of Amebiasis," delivered before the American Gastroenterological Association in 1944.

INCIDENCE

Amebiasis incidence reports have always been remarkably variable. In obviously well conducted studies emanating from military sources we encounter figures as high as 31.5 percent in America soldiers in and around Calcutta, India;[2] a modest 11 per cent involvement in soldiers who had been interned by Japs[3] and a low figure of 5.55 per cent in naval personnel.[4]

New York civilian population was granted only a .046 per cent amebic incidence In Chicago, a study of 4,605 cases revealed a comparative low figure of 0.7 per cent amebiasis.[6] Snorf and his associates[7] proffer the opinion that returning veterans and prisoners of war have not affected the incidence of amebic infection to date in an intensive study showing 2 per cent involvement in asymptomatic patients and 4.3 per cent in symptomatic patients. It is, however, obvious that publications on amebiasis are emanating from areas of our country where previously the disease was unrecognized and in this sense our premise was correct.

One must in reviewing some publications carefully weigh the circumstances surrounding the study. Illustrative is the excellent study by Thomas J. Mackie and his associates in Winston-Salem, North Carolina, showing a 31.6 per cent incidence of *Endamoeba histolytica* in veteran patients. But this was a specialized study at the Tropical Disease Clinic where an average of 5.2 stools were done per patient on individuals referred in with a service-connected disability, many already suspected of and already diagnosed as having amebiasis.

In our own experience, however, we are

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From the Browne-McHardy Medical Group. This study was supported in part by a grant from the Medical Research Foundation.

inclined to retract our warning of 1944 for our locality. In the 3,984 ambulatory patients surveyed as of 1943 we reported a 14.1 per cent incidence of amebiasis. In the past twelve months a study under identical circumstances showed a 12.4 per cent involvement in 1,547 case studies. Hospitalization studies in 1943 showed 5.2 per cent amebiasis, 2.08 per cent the second series.

A recent survey at Ochsner Clinic revealed a 9.9 per cent incidence of amebiasis in 1,330 patients carefully checked for parasitization.

At Charity Hospital the department of pathology shows no remarkable variation in incidence to indicate any startling change in the prevalence of amebiasis.

TABLE 1
INCIDENCE OF AMEBIASIS
CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS

Year	No. Studies*	% Amebiasis
1943	4,108	2.6
1944	3,447	4.4
1945	4,262	3.7
1946	4,162	5.6
1947	7,630	3.2
1948	10,105	3.2

*Data based on number of stools and not on survey of number of patients.

In its entirety this review does not justify the publicity given startling incidence approximations in excess of fifty per cent.

Craig's[8] conservative ten per cent amebiasis approximation for the United States contrasted with Faust's[9] "maybe as high as 20 per cent," coupled with our personal observation, leads us to feel that our incidence figure is quite accurate. In doing so we are certainly not minimizing the importance of frequency of the parasitization with a report of 12.4 per cent amebiasis for our 1949 study.

PUBLIC HEALTH PROBLEM

We adhere to the conclusion that parasitization with *Endameba histolytica* is the definition of amebiasis and that as such it is a significant clinical entity. Councilman and LaFleur,[10] in 1891, indicated the error of considering as innocuous those classified as being of the "carrier state." In 1917 Bartlett[11] conclusively demonstrated the pathology of amebiasis in persons dying of other disease entities and laid the background for the subsequent study. We concur in the judgement of Greenway and Castex[12] that the carrier state is a significant entity. It is no longer pardonable to consider the asymptomatic phase of amebiasis as an innocuous carrier state since extensive lesions in the intestinal tract and elsewhere may exist without producing striking symptoms or positive evidence of their presence. The inference is, therefore, that all patients found parasitized with *Endamoeba histolytica* should be properly treated, regardless of quiescence, until the parasite is permanently eradicated.

The public health problem recognized and studied in detail appears almost unsurmountable. Sanitation and personal hygiene are the most important issues. Detailed stool survey of all food handlers has been considered an impossibility but should be approached logically. Frequent surveys could possibly solve the problem. Unquestionably pre-employment and annual examination would be helpful. In 100 instances of chronic amebic dysentery we encountered 8 restaurant workers, 4 nurses, 2 dentists, 36 housewives, and 2 household domestics. This would indicate a high percentage of potential source of a communicable disease. Entries to this country from endemic areas should and could be required to have an adequate study. In 36 patients who came to us from tropical countries and 64 former military personnel who had either Pacific or North African assignments (to bring the figure to a hundred studies) the incidence of amebiasis was 19 per cent.

Little can be gained by the complete indifference to the public health we at present tolerate.

SYMPTOMATOLOGY

Amebiasis shows a symptom complex of highly diverse and changeable character, ranging in degrees of severity from attacks of acute fulminating colitis to all but negligible forms of bowel derangement. It has been conservatively estimated that over 50 per cent of parasitized individuals present

no manifestations specifically interpretable as indicative of amebiasis. The metastatic amebic involvements manifest themselves in relation to the site and extensiveness of the focus. In this respect we must not conclude that amebiasis is the most likely diagnosis in an instance of chronic diarrhea. Appreciative that no small series would be conclusive, that section, seasonal, economic, and age are important considerations, we nevertheless wished to depict the possibilities in our study.

TABLE 2
DIAGNOSIS IN 100 CASES OF CHRONIC DIARRHEA IN WINTER MONTHS

Irritable colon	34%
Diverticulosis	21%
Amebiasis	18%
Chronic ulcerative colitis	9%
Gastrogenous diarrhea	5%
Malignancy of colon	3%
Other enteric parasites	3%
Chronic pancreatitis	2%
Regional Enteritis	2%
Hyperthyroidism	1%
Benign colon polyposis	1%
Carcinoma of pancreas	1%

TABLE 3
DIAGNOSIS IN 100 CASES OF CHRONIC DIARRHEA IN SUMMER MONTHS

Irritable colon	19%
Diverticulosis	17%
Amebiasis	17%
Chronic ulcerative colitis	10%
Gastrogenous diarrhea	10%
Other enteric parasites	8%
Regional enteritis	5%
Chronic pancreatitis	4%
Malignancy of colon	3%
Ileocecal tuberculosis	2%
Endometriosis	1%
Lymphogranuloma venereum	1%

These patients, in a five-year follow-up, indicate no reason to change the initial diagnosis recorded.

Further, in our 1944 statistical survey of 790 cases of amebiasis we encountered 108 patients with amebiasis who had committant enteric disease producing diarrhea.

We must not, therefore, too glibly accept a diagnosis of chronic diarrhea due to amebiasis on presumption or on absolute laboratory demonstration of *Endamoeba histolytica* unless an adequate diagnostic survey has eliminated other etiologic possibilities.

TABLE 4
ENTERIC DISEASE CONCOMITANT WITH AMEBIASIS IN SERIES

Malignancy of colon	6
Benign polyposis	2
Proctitis, (non-specific)	11
Chronic idiopathic ulcerative colitis	4
Gonorrheal proctitis	2
Staphylococcal ulcerative colitis	1
Diverticulosis of colon	6
Chronic cecal volvulus	1
Endometriosis (serosal implantation)	2
Fistula in ano	3
Anal fissure	8
Marked anal constriction with recurrent impaction	14
Lymphogranuloma venereum	1
Bacillary dysentery infection	8
Other enteric parasites	28
Ileocecal tuberculosis	1

Of the last 16 instances of adenocarcinoma of the colon resected under our supervision 11 had been treated for amebiasis for an average period of five months.

Of the last 50 cases of irritable colon surveyed, 38 had been treated for amebiasis for an average period of twenty-six months. Among these were 11 patients who had been on antiamebic therapy for over thirty-six months without symptomatic response with the history of having been re-examined within ten days prior to our examination and told they were still parasitized. In these patients a more than meticulous survey by co-operative study with other laboratories failed to confirm the presence of amebiasis. Further, therapeutic response was prompt to release from amebaphobia and symptomatic measures. Many symptoms have been considered due to amebiasis; unless these respond to eradication of the parasite or be substantiated in a control series, their actual significance is doubtful. Miller[13] made a control study in children with amebiasis which is pertinent in its conclusions.

The inference: be a clinician not an amebiologist; evaluate all patients properly, consider amebiasis a clinical entity when parasitization is present but be cognizant

that it may be merely incidental to the patient's primary disease.

DIAGNOSIS

"No diagnosis of this infection can be said to be accurate unless it is based upon the demonstration of *Endameba histolytica* in the feces, exudates, or tissues of the infected individual." (Craig).[8]

Adequate parasitologic training, facilities, and saline induced fluid defecations (preferably a series of four) properly collected, proctoscopically checked, and immediately examined, facilitate the diagnosis. Zinc sulfate concentration floatation, staining technics, and especially the use of supravital stains, supplementary culturing and substantiating iron hematoxylin staining complete the picture.

At times a presumptive clinical diagnosis preferably aided by roentgen findings is confirmed by therapeutic response or by surgical demonstration of amebic pathology.

While not practical in all instances, Nelson's[14] alcoholic extract medium seems applicable to difficult diagnostic problems and to confirm curative claims.

COMPLICATIONS

It is startling to have patients present the fear of amebic brain and liver abscess imbued in them by a previous physician. It is alarming to read a published statement that amebic hepatitis occurs in more than 50 per cent of the cases of amebic dysentery, and liver abscess is found in from 3 to 5 per cent of these.[15] Spellberg and Zivin[16] with the conclusion that there is an increased incidence of active, virulent amebiasis in World War II veterans report 15 hepatic complications in 58 cases. They encountered an amebic cerebellar abscess. While earlier publications showed a high incidence of hepatic involvement most of these statistics were derived from autopsy studies. An amebic fatality is now a rarity. In 232 cases with amebic dysentery a diagnosis of hepatitis was ventured in 6.4 per cent. Amebic hepatic abscess was proven in 2.1 per cent. In our 790 cases of amebiasis studied in 1944 there were 15 instances of suspected amebic hepatitis and 5 cases of amebic hepatic abscess. In our 1949 study there were 4 instances suspected of amebic hepatitis and 1 proven amebic hepatic abscess in 193 cases of amebic dysentery.

We are inclined to feel complications are less frequent because of improved diagnostic accuracy and more effective therapeusis.

A summarization of complications encountered is tabulated.

TABLE 5

43 AMEBIC COMPLICATIONS IN 983 INSTANCES OF AMEBIASIS

Appendicitis (Acute)	6
Prostatic abscess	1
Hepatitis	19
Hepatic abscess	6
Perforation of colon	1
Granuloma of colon	6
Subphrenic abscess	2
Pleural effusion	1
Massive hemorrhage from amebic ulcerative colitis*	1

*Only fatality in series.

TREATMENT

A therapeutic program should persist until the *Endameba histolytica* are permanently eradicated; by this we infer a twelve month posttherapeutic follow-up with negative stool surveys at three month intervals. There are now available therapeutic agents which when properly employed can accomplish this.

To be effective an amebacidal agent must be absorbable; the known trophozoite penetration coupled with metastatic potentialities negates therapeutic claims for preparations sponsored for their surface and measured penetration efficiency. There must also be a correlation of drug localization with activity in selecting an amebacidal agent. Emetine, chloroquine and the thioarsenites are considered the more efficient for extraintestinal amebiasis. The iodohydroxyquinolines and pentavalent arsonic acids while effective in intestinal infections are probably not absorbed sufficiently and are excreted too rapidly to accumulate and be of curative value in extraintestinal involvement. A dearth of rational thought exists in reports on value of an

agent against cysts as compared to that upon the trophozoite yet reports of such study continue to appear in the literature.[17]

Emetine, experimentally proved to concentrate in the liver, is thereby efficient in hepatic involvement.[18] Indicated in the control of severe amebic enteritis and of extraintestinal amebiasis its efficiency thereon compensates for its high toxicity[19] and its "curative" potentiality estimated to be less than 30 per cent. We conservatively advocate limiting emetine hydrochloride to a daily 1 grain subcutaneous dose for eight days. We know of unusual instances where it has been given in such daily dose for three hundred and sixty-five days without apparent toxicity and of other instances of severe reaction after only a few days of therapy.

We feel E.B.I. and other oral preparations of emetine are not worthy of consideration.

Chloroquine parallels the tissue distribution of emetine[18, 20, 21] in intestinal penetration, liver, and other organ concentration. With insignificant toxocity it apparently equals emetine's efficiency in intestinal involvement. The only toxic manifestations we have encountered have been vertigo, tinnitus, and ataxia concurrent in 4 patients, which cleared rapidly on withdrawal of the drug and permitted restarting the medication in seventy-two hours. Headaches, visual disturbance, pruritis and gastrointestinal complaints have been reported.[21-24]

Proportionally superior hepatic localization,[25] greater amebacidal efficiency and less toxicity[26] of the thioarsenites over the pentavalent arsonic acids led to their clinical trial and conclusion of therapeutic efficiency in amebic hepatitis.[27] We would like to indicate that since arsenic is itself a hepatotoxic agent its use in amebic hepatic involvement may be contraindicated. We do not use any arsenical amebacidal agent when demonstrable hepatic disease exists.

Each of these three preparations advocated for use in extraintestinal amebiasis is acknowledged ineffective in intestinal parasitization. Therefore adjunctive amebacidal agents are necessary for the complete management of amebiasis.

Diodoquin, chinofon (yatren, anayodin), vioform and carbarsone are accepted therapeutically efficient for intestinal amebiasis. Their curative efficacy is reported to vary from 70 to 90 per cent for intestinal infections. The use of these agents alternately and in a wide variety of combinations probably compensates for the deficiencies of each and enhances the curative potentiality of the group.

A study of chiniofon metabolism[28] shows its absorption though rapid to approximate only 12.9 per cent and thereby indicates its limitation to intestinal amebiasis. This study is applicable to all of the iodohydroxyquinolines and to the pentavalent arsonic acids. Specific dosage is established. We do not favor the use of enemata prepared from this group feeling oral administration is satisfactory.

It is appreciated that herewith we have not covered all of the arsenical and iodohydroxyquinolines and their combinations that have been tried.

Bismuth glycolylarsanilate (Milibis, Wia., Win. 1011) is a more recently introduced apparently effective combination of 15 per cent arsenic and 42 per cent bismuth. Experimentally of minimal toxicity and maximal intestinal concentration explained by its insolubility it gives a wider margin of safety than other arsenicals with a greater intestinal efficiency.[29, 30] The advocated dosage is 0.50 gm. three times a day for seven days. As yet there have been no reported instances of arsenic sensitivity. We, however, have observed a single instance of arsenical encephalitis following excessive dosage in a patient suspected of being an alcoholic. (Berberian[31] in questioning this complication questioned its occurrence in a person not imbibing alcohol at the time). We have also observed an instance of typical mild exfoliate dermatitis occurring concomitant with the administration of milibis which cleared in sixteen days to recur when milibis was restarted. This second patient had not used alcohol.

The preparation apparently deserves continued trial. Winthrop-Stearns report a study of 461 patients with 82.6 per cent "cured."

During the past few years there have been many therapeutic agents advocated with a concept of amebacidal specificity but which appear, like the sulfonamides and vaccines, to be merely adjunctive. To this end many of the pharmaceutical laboratories have under trial combinations of antibiotics and amebacides.

Para-aminobenzoic acid gave symptomatic response.[32] Bacitracin has been granted a durative percentage.[33] Subtilin by surface tension lowering action is reported to rupture the amebic cell wall.[34] Aureomycin[35] has been a successful agent in 3 cases. The claims for penicillin and streptomycin have been conservative;[36] there is one report of prolongation of viability of ameba cultures by streptomycin.[37] The use of vaccines on the presumption of concomitant bacterial infection with Shigellosis and brucellosis[38] is mentioned.

At present we have under trial a less thoroughly absorbed chloroquine (aralen naphthoate) which may be more efficient for intestinal parasitization while adequate for extraintestinal involvement.

We are not concerned in this presentation with the surgical management of the complications. It is probable that with more adequate stool study, amebic consciousness and accurate application of proper amebacidal therapy together with further advances in chemotherapy the need for surgery may become rare indeed.

CONCLUSIONS

1. Subsequent surveys have failed to confirm our apprehension that amebiasis and other tropical diseases would become a pressing problem in the United States following World War II.

2. Publications and surveys have served to increase the alertness of the clinician, and reports and studies have become more widespread.

3. There is a definite Public Health problem which should be approached rationally, but the facts do not justify serious apprehension and the profession should guard against the development of "Amebaphobia."

4. Complications constitute only a small percentage. Possibilities and alertness in their management should be stressed, but certainly the exception not made the rule for approach to the problem of amebiasis.

5. Present therapy when properly applied shows a high degree of efficiency, and notable advances have been made in the management of extraintestinal infections.

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NOTE: Since the presentation of this paper we have a twelve week follow-up on 25 patients treated with aralen naphthoate. All 25 patients are entirely asympatomatic and a series of stool surveys have proved adequately the eradication of amebiasis. We do feel that this is an inadequate period of posttherapeutic follow-up and will continue this study. At the present moment, however, we are favorably impressed with the use of this drug as an amebacidal agent, in intestinal amebiasis.

HEART COMPLICATIONS IN BACILLARY DYSENTERY

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As early as 1930 one of us (D.N.S.) was the first to recognize the damage to the myocardium and the reaction of the sinus node mechanism to the presence of *B. dysenteriae*. In 1935, these findings were further corroborated by Silverman and Efron,[1] who were able to determine the allergic nature of these phenomena, as being due to hypersensitivity to the infection in some cases, while in others the same reaction followed the injection of small doses of concentrated autogenous vaccine. These changes showed all three types of sensitivity, local, focal and systemic, there being in all anaphylactic skin reactions at the point of injection; while in some there were vasomotor disturbances, arthritis, and even intestinal bleeding, due to vascular allergy. It is with these allergic phenomena and the toxic infectious lesions of the heart muscle that we are primarily concerned and are describing later in this paper.

The hearts of dysentery patients do not present any special symptoms. There is no cyanosis, and other symptoms of cardiac insufficiency are likewise absent.[2] The findings of Schittenhelm[2] are confirmed by those of Habs.[2] Gertenberg[2] (quoted by Habs), also states that enlargement of the heart and visceral congestion are not encountered. Late cardiac sequelae are very exceptional. No important lesions of the heart are observed in connection with the dysenteriae rheumatism in accordance with the observation that gravely ill patients who recover from circulatory insufficiency hardly ever present symptoms of heart lesions at a later date. It also seems that the heart suffers very little damage in dysentery, considering the seriousness of the disease. Uhlenbrach, (quoted by Habs),[2] states that he saw a considerable number of cases of myocarditis in the last year.

*Presented at meeting of the Orleans Parish Medical Society, October 10, 1949.

According to Michel,[2] (quoted by Habs), the dysenteric heart presents three types of muscle change: first, simple degenerative processes of the muscle, toxic cases that end quickly; second, interstitial myocarditis, such as may occur in the infectious disease; and, finally, only in a certain group of cases first observed in rheumatism and later observed in dysentery by Silverman.[1] The latter's cases had their origin in allergic reaction. Then others encountered tuberculosis, dysentery, scarlet fever, and similar diseases, viz., swelling of connective tissue near the vessels and Aschoff's granulomata. These hearts presented no symptoms when examined by auscultation, percussion, and x-rays.

Of 25 cases presented by Habs,[2] 15 showed sinus bradycardia, with only 1 having pathological changes in the electrocardiogram. In our series of 3 cases, none presented bradycardia, but all 3 had sinus tachycardia. There were pathological changes confirmed by electrocardiographic findings in 2 of 3 cases. In all of Habs's cases[2] the tachycardia and bradycardia were observed in the first week of the disease. We cannot definitely ascertain the date of onset of the lesions in our cases as in 2 of the 3, the lesions were present when the patients were first examined. In these cases the symptoms had been present for several days. In the third, there was a history of heart symptoms intermittently over an eight year period.

In the series of cases presented by Habs the pathogenesis of the changes in the electrocardiogram cannot be differentiated in a detailed manner. But our continuous observation does not admit of any doubt that the changes are caused by toxic infectious lesions of the heart muscle. We believe that according to the pathological research of Krauspe, the rare cases of quickly dying, toxic dysentery patients sometimes show degenerative processes in the heart, while at other times the cause is hyperergic inflammation in the sense of Kringe. But in most cases the changes are probably caused by interstitial myocarditis. It is, however, not possible clinically to distinguish the inflammatory changes from the degenerative ones.

The high percentage of pathological changes in the electrocardiogram leads to revision of the prevailing theory on the heart of the dysentery patient. Toxic infectious lesions of the heart muscle, mostly of a temporary nature, are by no means so exceptional as generally believed until now.

Herewith are presented 3 cases of chronic bacillary dysentery illustrating the toxic infectious lesions described by Habs,[2] and Michel,[2] 1 case in which permanent myocardial damage is present and the other 2 myocarditis of a temporary damage. It is our belief that the first named is one of interstitial myocarditis which is now evident even though the intestinal contents are free of *B. dysenteriae*. The other 2 show reversion back to normal of the electrocardiographs and other cardiac findings.

CASE REPORTS

Case No. 1. Mrs. L. E. T., admitted to Southern Baptist Hospital on March 9, 1949, after giving a history of loose bowels and mucus for six weeks. The loose bowels were associated with cramping. Although previous injections of emetine, and sulfadiazine by mouth had been administered, the diarrhea persisted and patient became progressively weaker. Stool cultures made on March 15, 1949, showed the Duval-Sonné strain of *B. dysenteriae*. The patient's serum gave positive homologous agglutinations in dilutions up to 1:640. At the time of admission, severe cardiac embarrassment, as manifested by sinus tachycardia, rate 120-160, dyspnea, and gallop rhythm, was present. A few days later a precordial friction rub and a protodiastolic murmur appeared. An electrocardiogram made at this time revealed depression of the S-T segments in all leads except lead III.

Cultures made on March 16, 24, and June 6, 1949, showed no evidence of Duval-Sonné strain of *B. dysenteriae*. An electrocardiogram made on June 4, 1949, revealed a normal rate and sinus rhythm with some reversion of the S-T segments towards normal but suggestive evidence of myocardial damage. There are periods at present when the rate will become rapid but no evidence is present on auscultation of the murmur described above, the gallop rhythm, or the pericardial friction rub.

Case No. 2. Dr. H. W. B., admitted to Southern Baptist Hospital, March 7, 1948, with chief complaint of diarrhea and fibrillation. History was that weeks previously he had fibrillation, took digitoxin; with fibrillation, he had apprehension

but no pain. Fibrillation would last a few minutes, then would be followed by tachycardia, a rate of from 140-160 for several hours. Upon further questioning it was revealed that this patient had dysentery while in the Service, in November 1944. An electrocardiogram made then had shown evidence of auricular fibrillation. He had episodes of diarhhea with cramps whenever he had this arhythmia. Cultures made on March 8 and 10, 1948, were positive for the Duval-Sonné strain of *B. dysenteriae*. Although the electrocardiogram made on March 8, 1948, did not show auricular fibrillation there was suggestive evidence of myocardial damage as evidenced by a Q3, T3 pattern.

He was started on the specific vaccine of Duval-Sonné beginning with dilutions of 1:100,000. Seven months later an electrocardiogram was made and the findings were normal; the arrhythmia previously reported had disappeared. Cultures were negative.

Case No. 3. Mrs. P. L. T., a white female, 47 years of age, gave a history that she had been treated by another physician during a period of eight years for intestinal autointoxication associated with paroxysmal tachycardia at infrequent intervals. For periods of several months she would be completely well then would develop diarrhea, fever, and pain in the upper left abdomen with a feeling of extreme weakness, and palpitation. At such times, the doctor usually found a very rapid heart rate ranging from 160-180. As soon as the fever disappeared, the heart rate dropped to normal. On one occasion, July 1921, at the beginning of these attacks, patient was hospitalized for over a month, and treated by colonic flushes, laxatives, and smooth diet with a minimum of roughage.

This patient was referred to one of us (D.N.S.) August 5, 1929, because of the recurrence of diarrhea and cardiac embarrassment for eight years. The history revealed that she had had an intestinal disturbance for two weeks. At the onset there was an acute episode of loose bowels. There had been similar attacks over a period of many years. During these many years her heart had given her much trouble, necessitating complete rest from time to time. Examination revealed a well nourished well developed, white female who showed certain abnormal findings. There were palpable posterior cervical glands and an enlarged palpable liver. The cecum apparently was full, and the sigmoid was palpable. A proctoscopic examination on August 5, 1929, revealed several deep ulcers in the rectum about 5 inches above the anus. There were several other superficial scattered ulcers. Agglutination tests with the patient's serum, made on August 12, 1929, showed strongly positive reactions with the Flexner strain of *B. dysenteriae*.

Desensitization, or so-called neutralization therapy, with dilution of autogenous vaccine was instituted with disappearance of the lesions in the rectum by February 2, 1930, and a disappearance of cardiac symptoms right after treatment was started September 1929. Heart rate returned to normal permanently and there was no recurrence of paroxysmal tachycardia after that.

DISCUSSION

Felsen[2] merely mentions the possibility of cardiac disturbance in acute bacillary dysentery; he makes no mention of its occurrence in the chronic disease, nor does he cite any concrete evidence of this combination.

Three cases of chronic bacillary dysentery in which the infecting organism,—two different strains of the dysentery bacillus,—has been instrumental in producing cardiac irregularities are herein reported. In view of the previously reported work of Silverman and Efron,[1] describing the many allergic manifestations of *B. dysenteriae* infection, principally including these of a vasomotor imbalance which could be precipitated by the administration of *B. dysenteriae* vaccine, it is thought the heart disturbances likewise brought out in this paper represent one of these allergic reactions. Then again the studies of the authors have shown a complete disappearance of cardiac symptoms following the eradication of the dysentery bacilli from the intestine of these patients. While this was true in all 3 patients, 2 showed complete reversion of electrocardiographic findings to normal.

SUMMARY AND CONCLUSIONS

1. To our knowledge, the first instances of heart complications incident to chronic bacillary dysentery encountered and studied in America are reported in this communication.

2. The authors feel that attention to the heart should be a routine study in *B. dysenteriae* infections.

3. All cases of chronic bacillary dysentery should have skin tests to determine the sensitivity of the patient to the infecting organism, since the avoidance of undue allergic reactions is predicated upon the concentration and dose of the vaccine.

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CONGENITAL FAMILIAL HEMOLYTIC JAUNDICE*

ISIDORE COHN, M. D.†
NEW ORLEANS

This presentation will be limited to a discussion of experiences with the congenital type of hemolytic jaundice.

It is essential for one who undertakes to do a splenectomy for hemolytic jaundice to bear in mind the necessity for accurate diagnosis in order to avoid the pitfalls which might arise from operating upon some of the acquired cases of hemolytic jaundice. It is true that some types of acquired hemolytic jaundice do respond well to splenectomy, but it is also important to remember that the familial type gives the best and most lasting results following splenectomy.

It is essential that there be close cooperation with internists, and hematologists, when one undertakes to establish a diagnosis and the indications for splenectomy in a case of hemolytic jaundice.

Hemolytic jaundice of the congenital type has been observed in this group, from the first to the fourth decade of life. Jaundice of varying severity, secondary anemia, and splenomegaly, are at times the only clinical manifestations. The laboratory furnishes the essential diagnostic characteristics of the disease. The characteristic red cells found in smears of hemolytic jaundice are spherocytes of the microcytic type. This abnormal form of red cell may constitute anywhere from 10 to 25 per cent of the total number of red cells. There is usually an increase in reticulocytes and an increase in the fragility of the red cells.

There are several questions which arise when one contemplates splenectomy for congenital hemolytic jaundice:

1. Why should one expect splenectomy to effect a cure?

2. What is the role of the spleen in the active phase of the disease?

3. Are spherocytes formed in the hyperactive bone marrow, or are they formed after leaving the bone marrow?

4. After splenectomy, even though spherocytes persist in the peripheral circulation, why is it that the hemolytic process does not recur?

These and many other questions have been investigated within recent years, and like many other questions associated with the functions and dysfunctions of the spleen, remain controversial.

It is accepted that the microcytic spherocyte is the characteristic cell found in hemolytic jaundice of the congenital type. Various theories have been postulated with reference to the origin of this cell which carries a stigma of increased fragility.

Naegeli, Haden, Fowler, and others consider spherocytes to be the result of specific bone marrow defects. Dameshek and his associates found that the immature red cells in bone marrow are of normal size, although the mature red cells in the blood stream are microcytic. They conclude that the abnormal cells are not formed in marrow but are the result of the action of some agent which has already reached the general circulation. They believe that spherocytes are formed outside of bone marrow from mature red cells by the action of various hemolytic agents. The exact site of the formation of hemolysin is not definitely known.

Dameshek in his latest contribution states that "The role of the spleen in normal and increased hemolysis is still obscure. The spleen is certainly an aid in most hemolytic processes and at times initiates and carries through the entire reaction."

He does not believe that a single mechanism can be considered responsible for all hemolytic states. As a result of his experimental work he believes: "That the vital activities of certain cells, notably those in the spleen, facilitate hemolysis."

It is concluded that a hypersplenism ex-

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ists and in these cases splenic activities may be responsible for increased hemolysis and the removal of the spleen results in a cessation of the hemolytic process.

The incrimination of splenic activity is accepted by most authorities.

Ham and Castle introduced a rather interesting hypothesis. They believe that an erythrostasis within the spleen is responsible for increased hemolysis. This idea is rejected by Dameshek and his associates.

Whether we accept the idea that increased destruction of red cells in the spleen is due to erythrostasis or to hypersplenism, it remains a fact that removal of the spleen does effect a remarkable clinical cure. The continued presence of spherocytes indicates that there is a congenital defect which is responsible for the presence of spherocytes of the microcytic type in circulating blood.

Enough has been said to be able to answer the questions which were proposed in the earlier part of the paper.

On theoretical and experimental grounds, one should expect splenectomy to effect a cure, because the spleen as a result of hypersplenism provides a more active hemolytic action on the already susceptible spherocyte. While it is true that the spleen may not be the entire factor, it does represent the major portion of the reticuloendothelial system responsible for destruction of spherocytes and the liberation of increased amounts of serum bilirubin, more than can be ordinarily removed from the liver.

Even though spherocytes persist after splenectomy the absence of exaggerated action of the spleen in the destructive process allows the patient to go on without marked evidence of increased hemolysis.

The diagnosis of congenital familial hemolytic jaundice rests on:

1. A familial history (not always obtainable).

2. The presence of jaundice, which may or may not be associated with abdominal pain, but which is not associated with the manifestations found in obstructive jaundice.

3. The presence of spherocytes in large numbers, increased reticulocytes, increased fragility of the red cells, increased serum bilirubin, and the finding of urobilin in the urine.

4. Splenomegaly.

This report is primarily based on personal experiences with a single family, five members of which I have operated upon at Touro Infirmary. The five cases represent two generations. In the first generation, there were two sisters and one brother. The second generation is represented by two daughters of the brother. The age of the patients varied from two years to thirty-four.

CASE REPORTS

Case No. 1. Mrs. H., age 28, first admitted to the Medical Service of Touro Infirmary, October 29, 1941. The patient stated that her father, one brother, and a sister suffered with the same condition.

The chief complaint was jaundice, weakness, and numbness of the extremities. She stated that she had had repeated attacks of jaundice, with spontaneous remissions for many year. She had been told that she had an enlarged spleen at the age of nine, and that the doctor had advised splenectomy at that time.

The salient features of the examination were: Jaundice, and a palpably enlarged spleen and liver.

The essential laboratory data follow: Red count 4,750,000; hemoglobin 64 per cent. There was increased fragility. Hemolysis began at 46 and ended at 25. The control hemolysis began at 40 and ended at 46. The reticulocyte count was 11 per cent. The icteric index was 50. The urine showed a two plus urobilinogen. The pathologist did not report the presence of spherocytes.

On November 17, 1941, a splenectomy was done. An enlarged spleen weighing 675 gms. was removed. Two accessory spleens, approximately 3 cms. in diameter were also removed. The pathologist reported that the accessory spleens presented an identical picture with the primary spleen: "The histological picture is consistent with the clinical diagnosis, congenital hemolytic jaundice."

Recovery was uneventful. The patient has since been kept under observation for follow-up.

Six months after operation the patient had gained 18 pounds. The skin was not found to be jaundiced. At that time no spherocytes could be seen in the wet preparation.

In 1945, four years after the operation, the blood picture showed: Spherocytes 12 per cent; Reticulocytes .09 per cent; Icteric Index 5. There was an increased fragility of the red cells, identical with the preoperative findings.

Five years after the operation the patient complained of sharp, stabbing, epigastric pain, the onset of which was usually one to two hours after meals.

Gall bladder visualization was reported as suggestive of cholecystitis.

In July 1947, five and one-half years after the original splenectomy, a cholecystectomy was done. Numerous small calculi were found in the gall bladder. At this time many spherocytes were seen in the wet preparation. The last follow-up blood study was made in June 1949, at which time the report stated: Hemolysis is slightly increased. Icteric index 4, Reticulocytes .05 per cent. No report was made on the presence or absence of spherocytes.

This patient's general condition is excellent and she has had no further jaundice.

Case No. 2. Mrs. E. B., age 34, admitted to Touro, November 15, 1943. The patient stated that she had jaundice in childhood with episodes of increased intensity and regression. Her chief complaints were substernal pain, nausea and vomiting, and dizzness for the past month.

Examination revealed slight icteric tinge of the sclera and skin, and a palpably enlarged spleen. The liver was not palpably enlarged. The blood picture prior to operation folows: Red count, 3,290,000. Hemoglobin, 70 per cent. There was increased fragility. Hemolysis began at 50 and ended at 34. The control began at 42 and ended at 24. The pathologist did not report on the presence or absence of spherocytes.

Splenectomy was done November 24, 1943. The spleen weighed 526.8 grams, and measured 17 by 12½ by 5 cm.

The pathologist reported "Splenomegaly histologically compatible with congenital hemolytic icterus."

In 1945, two years after the operation, the patient was found to be in good health, there was no evidence of jaundice. At that time the icteric index was 7. Reticulocyte count was 1.6 and spherocytes reported 20 per cent. There was an increased fragility. Hemolysis began at 50 and ended at 36. In the control it began at 44 and ended at 30.

The patient was last seen in August 1947, at which time no jaundice was found. Her general health was excellent.

She has refused to come in for further laboratory studies.

Case No. 3. J. A. B., age 3 first seen November 20, 1943. The mother stated that the child had had an enlarged spleen for a long time, and that she had had repeated attacks of abdominal cramps and jaundice. Three days prior to coming under observation the child had had an episode of abdominal cramps and jaundice.

On admission to Touro, the child was jaundiced; there was marked tenderness in the left upper quadrant, and a large spleen could easily be palpated, as far down as two fingers above the crest of the ilium. The liver was not palpably enlarged.

The peripheral blood study at that time showed a reticulocyte count of 3.7 per cent. Spherocytes .5 per cent. Red cell count 3,080,000. There was an increased fragility. Hemolysis began at 50 and complete at 32. The icteric index was 40.

A splenectomy was done on November 26, 1943. A large spleen measuring 15 by 7 by 3½ cms. was removed.

Repeated follow-up blood studies, have always shown a persistence of the spherocytes varying from the report of "Slight spherocytosis to 7 per cent." All of the follow-up studies also indicated persistence of an increased fragility of the red cells.

Since operation the child's health has been excellent and she has had no recurrence of jaundice.

Case No. 4. E. B., Jr., age 27. Admitted to Touro, October 10, 1944.

Chief complaint was repeated episodes of jaundice, nausea, and vomiting.

He stated that several generations back in his family have been affected with the same symptoms.

The peripheral blood study prior to admission to the hospital showed spherocytes 10 per cent. Increased fragility. Hemolysis began at 48 and was complete at 30. The control began at 40 and complete at 32. The red count was 4,500,000. Hemoglobin 89 per cent.

Abdominal examination revealed a large spleen which occupied the entire left upper and lower quadrants.

Icteric index was 15.

A splenectomy was performed on October 25, 1944.

Repeated blood studies since operation have shown a persistence of spherocytes, varying from "A few spherocytes present to 8 per cent". The icteric index has varied betwen 5 and 6 per cent. The reticulocytes have varied from .02 per cent to .08 per cent.

At the last examination, in 1949, the serum bilirubin was .05 mgms., and the cephalin flocculation was negative.

This patient has remained well; he has had no further jaundice, weakness, or any other complaint.

Case No. 5. J. A. B., age 3, daughter of Case No. 4, E. B. This child was first seen when she was six week of age. She was observed because her father, one sister, and two aunts, had been operated upon for congenital familial hemolytic jaundice.

I was unable to feel an enlarged spleen at this time. There was no discoloration of the sclera.

A complete blood study was requested—this advice was not followed.

Eleven months later, examination was made and a reticulocyte count of 8 per cent was found. Three

per cent of the red cells resembled spherocytes. Red cell count was 3,600,000. Hemoglobin 60 per cent. There was an increased fragility.

At that time the spleen was palpably enlarged.

In April 1946, an enlarged spleen and liver were found and peripheral blood study and hospitalization were advised with a view to splenectomy being done. The parents had the child admitted but refused operation.

In June 1947, the child was again seen because of jaundice, diarrhea, and temperature as high as 104° F.

Reticulocyte count at that time was 19.6. Total red count was 2,930,000. Hemoglobin 57.5 per cent. An increased fragility was found.

The pathologist reported that he was unable to find evidence of spherocytosis.

On June 8, 1947, a splenectomy was done, and the pathologist reported: "Histologically compatible with congenital hemolytic anemia."

In May 1949, peripheral blood study showed a reticulocyte count of 4 per cent. Slight spherocytosis. Persistent increased fragility. Total red count 4,650,000. Hemoglobin 84 per cent. Cephalin flocculation was negative. Icteric index was 4.

The child's condition at the present time is excellent.

CONCLUSIONS

During the period of time considered in this report 6 other proven cases of congenital hemolytic jaundice have been found in the records of Touro Infirmary by Dr. Leon, the Resident in Surgery. Recovery has followed in all of the cases.

At a later period we expect to give a report on statistics from Touro and Charity Hospital.

1. It is important to differentiate acquired from congenital familial hemolytic jaundice.

2. The results following splenectomy in the congenital type are much more satisfactory than reported results from operations in the acquired hemolytic types of jaundice.

3. Following splenectomy the characteristic blood findings (microcytic spehrocytes) persist in the peripheral blood.

4. There is a persistence of increased fragility of red cells.

5. A brief discussion has been given of the various theories of the origin of spherocytes, and the cause of increased fragility of the red cells.

6. Five cases occurring in one family successfully operated on have been included in this report.

NOTE: I wish to thank Dr. Hertzog and Dr. Nadler of the Departments of Pathology and Biochemistry (of Touro Infirmary) for their cooperation in connection with the study of the blood and blood chemistry; I also wish to thank Dr. Leon, Resident in Surgery at Touro Infirmary for his cooperation.

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TRACHEOESOPHAGEAL FISTULA

WITH REPORT OF A CASE TREATED SUCCESSFULLY BY LIGATION AND END-TO-END ANASTOMOSIS*

H. REICHARD KAHLE, M. D.**

NEW ORLEANS

Tracheoesophageal fistula is a condition in which the outlook for survival has been completely reversed by recent advances in intrathoracic surgery. Although it was first described by Durston in 1670 and by Gibson in 1703, Plass, in 1919, was able to collect from the literature only 136 verified cases and up to 1939 no instance of survival was on record.[1, 2] In that year, working independently but using the same general plan of attack, Leven[3] and Ladd[4] each successfully ligated a fistula and formed an anterior thoracic esophagus.

This was an enormous advance, but it was not the solution of the problem. Lanman,[5] in 1936, was the first to employ end-to-end suture, which had been mentioned by Holmes,[6] in 1869, in an extremely pessimistic report, as a possible mode of treatment. Lanman's attempt did not succeed, but he was undeterred by his failure and in 1940, when he reported 30 deaths in 30 surgical cases at the Boston Children's Hospital, 4

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treated by direct anastomosis, he still insisted that end-to-end suture was the procedure of choice and that it would eventually be performed successfully. It was only a year later that Haight[2] fulfilled his prophecy and thus ushered in an era in which the surgical mortality of this formerly hopeless congenital anomaly has steadily improved. Bigger's[1] experience is indicative of what has been accomplished: Between 1941 and 1948, he performed 11 operations for tracheoesophageal fistula, 4 of them direct anastomoses, with 1 survival. In 1949, he reported 7 additional operations, 5 of them direct anastomoses, with survival in all 7 cases.

Our local results, unfortunately, do not yet begin to approach Bigger's brilliant record, or the similarly excellent records of such surgeons as Haight[7, 8] and Ladd and Swenson.[6] In the 26 cases recorded at Touro Infirmary and Charity Hospital of Louisiana at New Orleans between 1935 and December 1, 1949, there were only 2 survivals, both of them in cases treated by anastomosis. One of the survivals occurred in the 3 cases which I have personally operated on* and the other in a case operated on by Dr. Craig Herringman. I should hesitate to bring to the attention of this Society so poor a record if it were not for the more optimistic outlook now available to these unfortunate children when the anomaly is promptly recognized and when the fundamental surgical principle of closure of the fistula is promptly applied.

INCIDENCE

The reported cases of tracheoesophageal fistula give no hint of its true incidence: As long as it was regarded as beyond cure, it was chiefly ignored. The distribution of the 26 cases observed at the two New Orleans hospitals makes that quite clear. Only 1 case was recorded in 1935 and only 1 in 1940. Only 7 others were recorded up to 1944. But in the last five years of the fifteen year period surveyed, 17 cases were recorded. Gray-Turner regards the incidence as about that of harelip and cleft palate,[6] and Haight,[2] who also emphasizes that only recent figures are of value, reports an incidence of 1:2,196 newborn infants. At the Boston Children's Hospital, the figures for which are naturally highly selective, there were 82 cases in an eight year period.

Fifteen of the 26 New Orleans cases were treated at Charity Hospital, 4 of them in negro children, and 11 were treated at Touro Infirmary. This is probably a fair index of the racial incidence since Charity Hospital, except for a small negro institution, is the only hospital in the city which admits negro patients. Sixteen of the 26 children were male and 10 were female.

EMBRYOLOGY AND PATHOLOGY

The basic cause of tracheoesophageal fistula remains to be clarified, but the malformation itself is easy to understand. The respiratory anlage, it will be recalled, develops from the foregut in a very early embryologic stage, the trachea and esophagus at this time being a single tube. The imperfection apparently has its inception between the fourth and twelfth weeks of fetal life, when, because of some error in the growth of mesoderm which normally separates the lung bud from the esophagus in the 4 mm. stage, the communication between the two structures persists. The esophageal atresia is also easy to understand. At this same time in embryologic development the esophagus normally becomes occluded by a proliferation of the epithelial lining, the lumen being later reestablished by vacuolization. If vacuolization does not occur, atresia persists.

Of the several classifications of tracheoesophageal fistula which have been advanced, the most important are by Vogt[9] and Ladd,[4] Ladd's being the simpler and more logical. In type I defects the upper portion of the esophagus ends in a blind pouch in the region of the first or second dorsal vertebra and the lower segment begins as a similar blind pouch at the level of the fourth or fifth dorsal vertebra. In type

*Another of these patients, although premature and weighing only 3 pounds, lived for four days after operation and probably would have survived except for a technical error in the institution of drainage.

II defects the lower anomaly is the same as in type I but the upper segment ends in a fistulous tract which enters the trachea just above the bifurcation. In type III defects, which are by far the most common, the upper segment ends blindly, as in type I, and the lower is connected with the trachea, just above the bifurcation, by a fistulous tract. In type IV defects the fistulous tract of the lower segment enters the trachea at the carina, instead of just above the bifurcation. Otherwise the malformation is of the type III variety. In type V defects both upper and lower segments communicate with the trachea.

In the 26 cases observed at Touro Infirmary and Charity Hospital 15 defects were of the classical type III variety and 8 additional cases were variants of this type. In 5 of the 8 cases the communication was at the bifurcation or the carina, as in Ladd's type IV, and in 3 other cases the communication was with the bronchus instead of with the trachea. In 1 of the 3 remaining cases the esophagus was atretic and there was no fistula, this being an extremely uncommon malformation; 1 case was of the type V variety, with both upper and lower segments communicating with the trachea, and in 1 case the description does not make clear whether the anomaly was type III or type V. There was no instance in the series of Ladd's type II, in which only the upper esophageal segment communicates with the trachea.

DIAGNOSIS

The diagnosis of tracheoesophageal fistula, and of the type of the defect, is not difficult if only the possibility is kept in mind. An infant who presents excessive saliva or frothy mucus, who has choking spells in which he becomes cyanotic, and who regurgitates his feedings and water should immediately arouse suspicion and a catheter (#10 F.) should at once be passed into the esophagus to determine its patency. Gage,[10] in fact, suggests that this procedure be carried out on every newborn child. If the plan were universally adopted, repeated efforts to feed the child would not be made, and the aspiration pneumonia, which so frequently folows them and which adds so much to the mortality, would not occur.

If atresia is present, obstruction to the passage of the catheter will be encountered 10 or 12 cm. from the gum margins. The next step is a plain roentgenogram. If it is inconclusive, or if more exact information is desired, a small amount of lipiodol can be instilled through the catheter to outline the upper esophageal segment. Under no circumstances should barium be used. It is extremely irritating, it is invariably aspirated, and aspiration pneumonia will be induced or, if it is already present, will be exacerbated.

The interpretation of the films is not difficult. In type I there is seen a dilated blind pouch ending opposite the first or second thoracic vertebra. In type III a similar blind pouch is seen. In type I there is no air in the gastrointestinal tract and no lipiodol in the trachea unless it has been aspirated into it. In type II there is no air in the gastrointestinal tract but lipiodol is present in the trachea. In types III and IV there is air in the gastrointestinal tract but no lipiodol in the trachea unless it has been aspirated into it. In type V there is air in the gastrointestinal tract and lipiodol is found in the trachea. This information is of both diagnostic and therapeutic importance: Type III which is fortunately the commonest variety, is the most readily amenable to surgical treatment, while types IV and V also lend themselves well to anastomosis. Haight[2] has estimated that when air is seen in the gastrointestinal tract on roentgenography, as it is in all three of these types, anastomosis is possible in approximately 83 per cent of all cases, while it is possible in only 20 per cent of the cases in which air is not present.

The routine use of tracheoscopy, as recommended by Byron[11], would no doubt increase the proportion of correct preoperative diagnosis, but the risk of edema seems a heavy price to pay for the possible benefits.

The child should always be examined completely because of the likelihood that other anomalies will be present, particular-

ly imperforate anus. All series call attention to this possibility. Seven of the 26 patients treated at the two New Orleans hospitals presented associated anomalies. Two had horseshoe kidneys, 1 a diverticulum of the ileum, and 1 a bipartite uterus and vagina with a persistent cloaca. The 3 other patients in this group all had imperforate anus, in one case associated with dextrocardia and in another with agenesis of the gallbladder and possibly atresia of the bile ducts, though the autopsy protocol is not clear on this point. With the possible exception of the last case, none of the associated anomalies threatened life or, if they did, were beyond surgical correction.

OPERATION

A few children with small esophageal fistulas have survived for variable lengths of time,[8] but when atresia is present life is incompatible with it without surgical relief. Moreover, as Richter[12] emphasized in 1913, ligation of the fistula is essential. The long record of failure in the cases in which this step was omitted is clear indication of its necessity.

Some form of inhalation anesthesia is best for the patient and simplifies the procedure for the surgeon. In my last two cases cyclopropane was used, with excellent results.

The surgical approach depends upon the type of anomaly present, which determines, in turn, whether or not anastomosis is feasible. If it is, the incision of choice is usualby a right-sided extrapleural approach to the mediastinum. In the two cases in which I used a right-sided retropleural approach exposure was better than in the third case, in which the approach was from the left. In that case retraction of the heart was followed by cardiac disturbances and the arch of the aorta hampered manipulations. Lyon and Johnson[13] seem to be alone in recommending a transpleural approach.

Posterior resection of segments of the second, third and fourth ribs, and sometimes of the fifth rib also, is usually part of the procedure, though Bigger[1] has recently advocated a retropleural approach through the bed of the resected fourth rib. It is difficult to avoid damaging the pleura, in spite of careful manipulation.

It is frequently necessary to divide the azygos vein between ligatures, to permit visualization of the lower esophageal segment and its communication with the trachea, as well as to identify the vague nerve. The blind proximal segment of the esophagus is then sought for in the superior mediastinum. Sometimes it is necessary to insert a catheter before it can be located and mobilized. After the fistulous communication of the lower segment is ligated and divided as close to the trachea as possible, the upper pouch is opened, and the segments are anastomosed by simple end-to-end suture or by a telescoping technic. Technical difficulties are frequent because the lower segment is likely to be small and poorly developed while the upper segment is usually dilated and hypertrophied. Tension on the suture line must be avoided as far as possible, as it is usually followed by leakage, though the development of a fistulous tract is not necessarily fatal. The lung is then re-expanded and the wound is closed in layers, a small drain being left in the mediastinum. I am not in favor of leaving a catheter in the esophagus, as pressure at the anastomotic site may predispose to leakage.

Since oral feedings are contraindicated for twelve to fourteen days after operation, gastrostomy is a necessary part of the operative procedure. Ordinarily it is done twenty-four to forty-eight hours after the anastomosis, but Bigger[1] has recommended that it be done as the first phase of operation. I used this order of procedure in my own successful case, to relieve distention of the gastrointestinal tract and to secure more time for preparation, since the child was an extremely poor risk, but at the time Bigger's recommendation had not come to my attention. When oral feedings have been instituted successfully, the gastrostomy is permitted to close. The Stamm technic is usually employed.

If primary anastomosis is not possible, as it usually is not in types I and II of this anomaly, the choice of operation lies be-

tween the formidable transthoracic esophagogastrostomy suggested by Byron[11] and a staged procedure consisting of ligation of the fistula, upper (cervical) esophagostomy, lower (thoracic) esophagostomy, and after a considerable time, connection of the esophageal stomas by means of the simplified plastic procedure described by Lam.[14] Sweet, [15] in order to avoid the extensive plastic operations necessary in the construction of an anterior esophagus, has suggested that the stomach be mobilized through the thorax and anastomosed to a previously formed cervical esophagostomy in the cervical region.

PREOPERATIVE AND POSTOPERATIVE CARE

The prevention of aspiration pneumonia and the maintenance of a proper fluid balance are the chief considerations of preoperative preparation in a child which tracheoesophageal fistula. To prevent pneumonia, the head should be lowered and continuous suction or, better, repeated catheter aspirations should be carried out. If atelectasis or pneumonia is already present, it is well to adopt Haight's[7] suggestion and keep dependent the side on which the incision is to be made.

The correction of dehydration and the maintenance of a proper fluid balance are important both before and after operation, but fluids must be administered cautiously, to avoid the development of edema. Saline solutions are particularly likely to cause this complication, and it is probably better to err on the side of conservatism and limit fluid administration to dextrose solution and Ringer's solution. The administration of potassium is important, but in the absence of easily available methods of measuring potassium blood levels, great caution must be exercised in its use. Blood is usually not urgently needed in the preoperative period, but is essential in the postoperative period, since the loss at operation is frequently much greater than is realized.

Penicillin is given before operation, as a prophylactic measure against pulmonary infection which may develop, or as a therapeutic measure if it already exists. After operation it is continued, in the amount of 10,000 units every three hours. Other antibiotics and the sulfonamides are employed according to the indications.

The child is placed in an incubator and is given oxygen continuously. The pharynx is aspirated at intervals if mucus accumulates. Nothing is given by mouth for ten or twelve days after operation. Then, if primary healing has occurred, feedings are begun in gradually increasing amounts. If leakage has occurred at the suture line, they are withheld until the fistula closes.

There are few other conditions in which, both before and after operation, closer cooperation is necessary between the pediatrician and the surgeon. I am quite certain that the excellent attention of the pediatric residents had much to do with the recovery of the child in my own successful case, the history of which is appended, for the record.

CASE REPORT

A white male child was admitted to Charity Hospital of Louisiana at New Orleans, January 11, 1949, on the tenth day of life, with a history of regurgitation with choking and cyanosis at every attempt to feed him or give him water. He had had no treatment except for a daily hypodermoclysis. Examination revealed a small, malnourished and greatly dehydrated infant, in poor physical condition, with signs of pneumonia in the right chest. A catheter passed into the esophagus encountered an obstruction at the level of the second rib anteriorly. A plain roentgenogram of the abdomen and chest confirmed the diagnosis of pneumonia and revealed gas in the stomach and intestines. A second roentgenogram, following the injection of lipiodol into the esophagus, showed that its upper portion ended in a blind pouch.

Preparation for the operation consisted of blood transfusions, penicillin by the intramuscular route, and the intravenous administration of 5 per cent dextrose solution, amigen, lactate-Ringer's solution, and sulfadiazine. Thirty-six hours after admission, when the child's initially poor condition had greatly improved, a Stamm gastrostomy was performed under local analgesia. The following day the tracheoesophageal fistula, which was of the type III variety, was ligated and an end-to-end anastomosis was performed. The right-sided retropleural approach was used, with ligation and division of the azygos vein. Drainage was instituted by means of a small rubber catheter. Operation was without incident except that the pleura was accidentally entered.

The postoperative regimen included blood transfusions, intramuscular injections of penicillin, and

intravenous infusions of dextrose solution. Feeding via the gastrostomy was begun the day after operation and oral feedings of a standard formula were begun eighteen days later. When the patient was discharged on the seventieth postoperative day he weighed over 8 pounds, approximately 3 pounds more than his preoperative weight. He was taking all his feedings, including cereals, by mouth without difficulty. The gastrostomy had been permitted to close.

The child has been seen in the clinic several times since his discharge. Ten months after operation he weighed 18 pounds but because his mother reported that he sometimes choked on solid foods he was hospitalized for observation. Examination of the esophagus with an opaque meal showed some narrowing at the site of anastomosis but no delay in the passage of the material. The nurses, who were instructed to watch him carefully, reported that he regurgitated his food only when he was emotionally upset at feeding times. An otolaryngologist saw the child in consultation and dilated the strictured area through an esophagoscope. A repetition of this measure may be necessary, though most observers are agreed that constrictions of surgical origin, unlike lye constrictions of the esophagus, seldom require more than one or two dilatations.

SUMMARY

Tracheoesophageal fistula, which was formerly a hopeless anomaly, is now amenable to surgical correction, by anastomosis of the esophagus and ligation of the fistula, with a steadily improving mortality. Prompt diagnosis and careful preoperative and postoperative management are essentials of success. The important clinical details of the condition have been outlined, and another successful case has been recorded.

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LUETIC AMYOTROPHIC MENINGOMYELITIS

CASE REPORT

H. THARP POSEY, M. D.

NEW ORLEANS

Kinnier Wilson says of this condition, "In respect of severity, evolution and site, lesions differ widely, and they are sometimes combined in such a way as to form links with other widely recognized types." Unfortunately, what with "practical" specifics for the treatment of syphilis of all forms in later years, little has been added to the modern commonly used neurological and medical texts to bring more diagnostic exactitude to a condition so variable in expression as Wilson has indicated, nor does the modern journal literature often mention this disorder. If Wilson's citing of workers and "authorities" of the last century and the first three decades of the present one today be granted the validity he attributed to them, Osler's dictum of the protean nature of syphilis is especially corroberated by spinal syphilis.

The following case is one in point. It is conceded that, if it be considered a *sine qua non* of the diagnosis, no *T. pallidum* were found.

From the Neuropsychiatric Section, VA Hospital, New Orleans, and the Department of Psychiatry, Tulane University School of Medicine. Published with the permission of the Deans' Committee of Tulane University and Louisiana State University.

CHIEF COMPLAINT AND PRESENT ILLNESS

This 58-year-old white male was admitted October 4, 1946 with a chief complaint of inability to walk or use his hands, massive progressive weight loss, and general debility. There was no psychiatric complaint.

The present illness was said to have begun in April, 1946, with intermittent claudication. Prior to this time, entirely good health was claimed. He complained of pain in his calves and feet, and progressive fatigue. The pains in the calves were described as "cramps, as if the leaders were shortened." The pains would disappear after rest following a day's work. In late April, the patient saw his company physician who prescribed vitamin tablets which gave no relief. In May, he was forced by his disabilities to cease work and he entered his company hospital for treatment. There he was told he had a urinary infection and he received five injections of penicillin. In addition, all his teeth were extracted. He was told his blood Wasserman was four-plus. He did not improve at all at that hospital and was referred to a private physician in Port Arthur, Texas. This physician performed a diagnostic lumbar puncture in the course of his care and took another blood Wasserman. The former was allegedly negative and the latter strongly positive. The physician placed the patient on weekly injections of bismuth and the patient took five before deciding he was getting worse and entering another hospital in August. Prior to this time, he had become totally unable to walk in early June due to weakness of both feet. During his thirteen-day stay in the private hospital, he received 7,800,000 units of penicillin in eight days, and heavy doses of B-complex. A blood serology on August 24, 1946 was reported negative (verified). Leaving the hospital Maximal Benefit, he convalesced at home. The disease progressed, however, and by mid-September he had lost the use of his hands. A month before admission to VA Hospital, New Orleans, he suffered an attack of transient dyspnea which cleared without any difficulty.

Review of systems was negative except for neurological difficulties.

PAST PERSONAL HISTORY

Patient gave a history of a penile chancre in 1914, for which he sought early treatment and was given four injections of salvarsan, after the second of which the lesion began to heal. He received no more antiluetic or other treatment. In general, his health was good until the onset of the present illness, except for falling in weight from over 215 pounds to 161 on admission. In 1944, he had had some "fatty tumors" excised from his left forearm without sequelae, and one year before admission here, he had had a transient swelling of the ankles which he attributed to "kidney trouble" and for which he took no treatment. His early medical history included the usual childhood diseases and typhoid, from each of which he recovered uneventfully.

The patient spent one year in the Army during World War I, saw no overseas service, earned no promotions, awards, and stood no courts-martial. After Army discharge, he was employed as a mechanic by the T. & P. Railroad and remained so until the present illness forced him to cease work. The marital history was negative and children living and well. The wife had no miscarriages. Patient was a consistently heavy smoker, drank only an occasional beer.

FAMILY HISTORY

Family history was noncontributory. The father had died of a "stroke," the mother of hypertension. For a number of years before her death, the mother had had a "crippling arthritis of the hands."

GENERAL PHYSICAL AND NEUROLOGICAL EXAMINATIONS

The positive findings on the general physical examination were: (1) weight 161, height 71 inches; (2) tonsils atrophic; (3) edentulous; (4) scattered fine rales over both bases posteriorly; (5) liver border 5 cms. below the costal margin; (6) prostate not examined; (7) moderately severe pitting edema of ankles and feet; (8) old pigmented circular scar, d-6 cms., an-

tero-medio-distal aspect left leg; (9) bilateral trichophytosis pedis; (10) no penile scar.

The positive findings on the neurological examination were: (1) hyperhidrosis of the palms; (2) moderately severe atrophy of the temporalis and masseter bilaterally; (3) moderately severe atrophy of anterior and posterior neck muscles bilaterally; (4) posture quadriplegic in flexion; (5) generalized atrophy of the muscles of the arms and forearms bilaterally but more pronounced in forearms; (6) bilateral severe atrophy of the first lumbrical with mild atrophy of the interossei and other lumbricals bilaterally; (7) bilateral severe quadriceps atrophy; (8) bilateral severe peroneus and gastrocnemius atrophy; (9) generalized hypotonus of all extremeties; (10) total paraplegia from below knees bilaterally. On active flexion of the thighs with marked effort, there was passive flexion of the legs; (11) bilateral wrist drop with total paralysis of the hands and fingers; (12) obvious fasciculations over both pectorales majores; (13) the upper and lower abdominal reflexes were present but sluggish. The cremasteric reflexes were active bilaterally. The biceps were present bilaterally but sluggish. The jaw jerk was active. All other reflexes were absent. There were no pathological reflexes; (14) there were no meningeal signs; (15) bladder function was subjectively normal; (16) rectal sphincter exhibited normal tone; (17) there was a generalized hypesthesia of the lower extremities bilaterally; (18) stereognosis was bilaterally absent in the hands but barognosis was present and normal; (19) there was hyperalgesia of deep sensation in both lower extremities; (20) there was an indefinite disorder of temperature sensibility over all extremities but in the forearms and hands primarily confined to difficulty in identifying warmth; (21) position sense in the hands and feet was completely disordered; (22) vibratory sense was markedly reduced over all extremities. Two-point sensibility was not tested.

LABORATORY FINDINGS

Complete blood counts: Oct. 7, 1946, RBC 4.63, WBC 8.2 with N.73, L.20, M.6, E.1; Dec. 11, 1946, RBC 4:38 with Hb.85, WBC 6.8, with N.64, L.32, M.2, E.2; Dec. 20, 1946, RBC 4.72 with Hb.95, WBC 9.95 with N.71, L.27, M.1, E.1.

Sed. rate Oct. 9, 1946: 30 mms.; Nov. 7, 6 mms.; Dec. 7, 12 mms.; Dec. 20, 12 mms.

The first five urinalyses shortly after admission showed variously traces of albumin and pus cells varying from a few WBC to gross pus; subsequent urinalyses entirely negative. Fishberg, Oct. 9, 1946: 1019, 1015 and 1020.

Total serum protein and A/G ratio: TP 6-2, A 3.9, G 2.3, A/G ratio 1.7.

Six separate blood Kahns and Kolmers were all positive.

Fasting blood sugar 95. NPN 42.

Complete spinal fluid survey in lateral position Oct. 7, 1946: IP 13 cms, TP 9 cms, 15 lymphocytes, Wasserman and Lange negative, total protein unobtained. Oct. 8, 1946, 5 lymphocytes, Wasserman and Lange negative, total protein 190 mgs. per cent. Oct. 14, 1946: 5 lymphocytes, Wasserman positive (44320), Lange 3445522100, total protein 207 mgs. per cent.

X-ray of the chest on admission revealed clear lung fields and normal cardiac silhouette but the left costophrenic angle was obliterated and there was evidence of old adhesions in the left leaf of the diaphragm. X-ray of skull on admission was negative. X-ray of the sinuses on admission showed minimal clouding of the left maxillary sinus. X-rays of both tibiae on admission showed a shadow of calcific density in the anterior soft tissue at the junction of the middle and proximal thirds of the left tibia. No exidence of bony pathology was noted.

Electrocardiogram Oct. 9, 1946, reported as "Strongly suggestive evidence of myocardial disease; may be compatible with chronic emphysema." The recording was reported by the technician as being poor, repeat was requested. Repeat electrocardiogram reported as "Low voltage QRS and T complexes may be due in part to

emphysema. Strongly suggestive evidence of myocardial ischemia in addition."

CONSULTATIONS

Consultation was had with the Medical Service for diagnosis and treatment of the medical condition. The consultant stated he was not familiar with the clinical picture presented but did not consider that further antisyphilitic therapy was indicated. A second consultation was requested with Medicine after the electrocardiographic report. The consultant was of the opinion that the cardiac and renal findings were simply incidental to the primary disease. Consultation was had with Urology because of the urinalysis findings and the history. Consultant reported evidence of mild prostatitis but considered the remaining symptoms as being compatible with chronic glomerulonephritis. He was of the opinion that no urological excretory lesion appeared evident at the time, and suggested study of the serum proteins. Consultation was had with Ophthalmology because the discs appeared slightly hazy and there seemed to be a slight temporal pallor OD. A consultant considered the findings physiologic. A small spindle shaped, dark area measuring .5 mms. by .1 mms. was found immediately superior to the disc OS along the superior temporal venule. Its significance was not established.

COURSE IN HOSPITAL

After the initial work-up had been completed, the case was diagnosed to be one of progressive muscular atrophy of an undetermined type by some, and by the writer a case of luetic amytrophic meningomyelitis. In view of the consistently positive blood serology and one positive CSF serology, treatment was begun on the latter assumption and the patient was given 8,000,000 units of penicillin and ten weekly injections of bismuth subsalicylate. In addition, he was given one ampule of Solu-B (lx) intramuscularly daily, and by mouth massive doses of ferrous sulphate, vitamins A and D, and ascorbic acid. Nineteen days after admission, having received 1,440,000 units of penicillin and one injection of bismuth, in addition to the massive vitamin therapy since admission, the patient stated that he was considerably improved and much stronger. He was truly quite alert and interested in his surroundings, laughed readily, ate well and slept well, but there was no other evidence of improvement. In fact, the staff was of the opinion that the atrophy, particularly of the face and neck, was progressing noticeably. Furthermore, there was no change in the edema of the feet and there was a consistent slow fall in weight. As noted under laboratory findings, the sedimentation rate had fallen from an initial high of 30 to 12 and the patient remained afebrile as on admission. After the patient had been given 8,000,000 units of penicillin, he was sent to physiotherapy every other day where he received passive exercise, radiant heat, and massage. After about a week of physiotherapy, the patient had regained the most minute use of his fingers and feet in that he could just flex the tips of his fingers enough to be perceptible and was able to initiate a movement of extension with both feet. It was thought that this return of function and marked subjective improvement was an indication that treatment was arresting the disease progress, yet every ward round led to comment that the patient's face was growing more and more gaunt day by day and one could not escape the impression of slight dypnea in spite of the fact that the patient denied any difficulty in breathing. On the fifty-fifth day of admission, and without apparent cause, the patient displayed an acute respiratory failure and it was necessary to place him in an iron lung. There were no immediate signs of cardiac embarrassment or pulmonary congestion. He spent almost two entire days in the apparatus and then was able to be removed from it for increasingly longer periods. After a week, he was able to be out altogether and remain comfortable. A week later he resumed physiotherapy and was frequently up in a wheelchair thereafter. He continued to insist that physiotherapy was strengthening his arms and legs although there was no evidence of this beyond that already described.

On the eighty-second hospital day, respiration became acutely rapid and the patient asked to be returned to the respirator which was done. By the time he was placed in the respirator, he was perspiring profusely, pulse was 116, respiration 28 and he was complaining of pain in the right lung. His condition grew rapidly worse from that time. Pulse, temperature, and respiration rose slowly but steadily and the physician and attendant were unable to synchronize the patient's respiration with the respirator. On the third day after being returned to the respirator, the patient became semi-stuporous and pulse varied between 120 and 140 and respiration between 20 and 30. Respirations were extremely labored. In the agonal state of the next ten hours, the pulse became almost uncountable, respiration markedly irregular and rapid and the temperature rose to 107 at the hour of death on the eighty-sixth hospital day.

Autopsy resulted in diagnoses of: (1) Myeloma, lymph nodes, hilar, (2) Cerebrospinal congestion and edema, (3) Bronchopneumonia, bilaterally, lower lobes, (4) Pulmonary abscesses, left lower lobe, (5) Mitral and aortic valve atherosclerosis, (6) Hepatomegaly, (7) Splenitis, septic, (8) Nephrosclerosis, benign, (9) Aortic atherosclerosis, (10) Prostatic hyperplasia, (11) Pleural adhesions, between parietal and visceral pleura, and between all lobes.

The specimens of the central nervous system were submitted to the Army Institute of Pathology where examination was done by Dr. Webb Haymaker who reported as follows:

"The most striking finding consisted of loss of nerve cells in anterior horns. The remaining cells were often shrunken or otherwise distorted. The distribution of cell loss varied a good deal, generally being most pronounced in the intermediate and lateral portions of the anterior horns. Occasional vessels in the anterior horns were surrounded by a few lymphocytes. Anterior roots showed considerable degeneration of myelin and axis cylinders, and the same was true of some of the posterior roots. The latter finding is borne out in one section from the cervical cord in which the Weil stain disclosed mild degeneration of myelin in the more medial port of the fasciculus gracilis. The case is regarded as one of syphilitic amyotrophy.

SUMMARY

A case of clinically diagnosed luetic amyotrophic meningomyelitis with histopathologic confirmation is reported.

NOTE: Thanks are due to Mr. William Gude, Department of Anatomy, Tulane University, for the variety and excellence of the special stains.

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A BURN INVOLVING EIGHTY PER CENT OF THE BODY WITH SURVIVAL

REPORT OF A CASE

WARREN L. ROSEN, M. D.

NEW ORLEANS

Few patients, if any, have survived a burn involving more than 65 per cent of the body surface. Pack[1] has stated that "all burns of the first degree are fatal if two-thirds of the body surface is involved, and all burns of the second degree are fatal if one-half of the body surface is involved." He also stated that "scalds are more serious area for area, and depth for depth than thermal traumas due to dry heat." This opinion has been shared by others such as Christopher[2], Mac Leod[3], and Lund[4]. I, herewith, wish to report a case of a scald with survival, that involved over 80 per cent of the body surface according to the accepted Berkow tables.[5]

REPORT OF A CASE

T. L., negro male, age 39, a worker in a large cotton mill, was first seen in the Emergency Room at Touro Infirmary at 4:30 AM, October 3, 1947. The history obtained was that he had been instructed to mix a large vat of dye and in so doing, the tank had exploded covering him with scalding water. Later investigation revealed that the proper procedure was to heat a large tank of water to boiling, start a mechanical agitator and add the powdered dye. He had heated the water to boiling point but forgotten to start the agitator so that when the powder was added it formed a solid film over the top of the tank of water causing the

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Senior Surgeon, Touro Infirmary.

water to boil over. He had been covered from head to foot with this boiling water and powdered dye and severely burned.

Examination and first aid: He was first seen by the intern on accident service, who did not realize the magnitude or seriousness of the burn, and seeing him covered with a blue dye, sent him with an orderly for a shower bath. I arrived a few moments later while he was in the shower. He had been stripped and it was noted that his entire body had been burned. (see Table I for chart of his burns). He was in mild shock, pulse rate 106 per minute, and blood pressure 100/60 mm., and he

TABLE I
BERKOW'S TABLE

	% Normal Area	Burned Area
Head	7	4
Neck	2	2
Chest	13	13
Back	13	13
Up. Arms	8	8
Forearms	6	6
Hands	5	5
Genitals	1	....
Buttocks	5	2
Thighs	19	14
Legs	14	10
Feet	7	4
	100%	81%

was complaining of severe pain. He was given morphine sulphate 1/4 gr. and an infusion of plasma was started. Because of the extensive burned surfaces and the collapse of the superficial veins it was necessary to cut down on one of the anterior tibial veins above the ankle to get the infusion started. No further attempt at cleansing the burns or any debridement was made and all the burned surfaces were covered with fine mesh gauze impregnated with vaseline. This was covered with dressings of five yard rolls, and slight pressure made with elastic Ace bandages. His neck, chest, abdomen, back, both arms, forearms, and hands, thighs, legs, and feet were similarly treated. His face, head, and eyes were also burned but no dressings were applied to these parts except a protective coating of plain vaseline. The dressings took about two hours to apply at which time he had received 500 cc. of plasma. He was then transferred to a private negro hospital in an ambulance.

Course in Hospital: On admission to the hospital, his temperature was 98°F., pulse rate 84 per minute, respiration 30 per minute, and blood pressure 110/60 mm. A hematocrit was reported as 52, so an additional 500 cc. of plasma was given following the rule of Harkins that 100 cc. of plasma should be used for each point the hematocrit is above normal. Besides this he was given 1000 cc. of 1 per cent saline in 5 per cent glucose. Penicillin was started in doses of 30,000 units every three hours and this was continued until October 28, at the time of his second dressing. He was encouraged to take fluids by mouth and received at least 3500 to 4000 cc. daily. Opiates were ordered to relieve pain but following his original dressing, he remained comfortable, and it was only necessary to give him three doses of codeine sulphate 1/2 gr. tablets, to relieve any discomfort during his hospital stay. A blood count made October 4, 1948, showed total red blood cells of 4,340,000 with hemoglobin of 95 per cent. White cell count was 10,450 with 74 per cent neutrophiles, 23 per cent lymphocytes, 3 per cent large mononuclears. Urinalysis showed specific gravity of 1.016 with no albumin, no sugar, and no red blood cells or casts in the sediment. A check of the urine was made daily during his hospital stay and at no time did it show any abnormality. The specific gravity remained constantly above 1.014 and there was never any albumin. A hemoglobin determination, made October 21, 1948, was 70 per cent, and red blood cells were 4.3 million.

He was seen in consultation by Dr. W. M. Boles for his eye condition, and this proved to be only superficial burns of both cornea which responded rapidly to treatment. He was kept on a high protein diet of 200 grams with 4000 calories, and ate well. It was never necessary to repeat infusions as he remained well hydrated and although we were never able to get plasma determinations, his general condition remained so satisfactory that we did not feel it necessary to supplement his feedings with extra protein or blood. On October 13, ten days following his injury, he was taken to the operating room and under sterile precautions was dressed. The Ace bandages had become loose by this time and had become uncomfortable. Without any anesthesia, the dressings were removed, the burned areas washed with soap and warm water, and then re-covered with fine gauze impregnated with vaseline and large dressings of gauze. These were then covered with new Ace bandages. There was evidence that all burned areas were healing, and only in a few places were there areas of deep granulation. When the original dressings were removed, all the superficial skin that contained black pigment came off with the gauze and almost his whole body was red instead of black. The burns of his face had almost healed during these ten days and this was the only part of the burned area that showed repigmentation. Unfortunately, photographs were not made at this time as the contrast was striking between the red areas of healing and the few original parts of black skin. He was not dressed again until October 28, twenty-five days following the burn, at which time photographs were made in the operating room (see attached photographs).

These photographs were made October 28, 1947, or twenty-five days folowing the burn. The face had completely healed by this time. The darker areas represent the only areas of skin which were not burned.

By this time, his face had completely healed and become fully pigmented again. The areas as shown in the photographs were still red although they showed some beginning pigmentation. Only a few small areas over his sacrum, shoulders, and elbows showed granulating areas. These were dressed and the patient was discharged on October 31, and told to report to the office for further treatment.

Progress: The small areas over his shoulder, elbows, and sacrum were dressed three times a week for the next four weeks and finally all areas healed without the need for grafting. There were no constricting scars and all joints were freely movable. After his discharge from the hospital, his diet was supplemented with high vitamin concentrate tablets and extra protein in the form of Protenum. He was allowed to return to his job January 5, 1948, about three months following his burn, at which time he was completely healed and well.

COMMENT

Of the patients admitted to the Massachusetts General Hospital for treatment of burns received in the Cocoanut Grove fire, only 1, (case No. 13), recovered following extensive burns. This patient had 56 per cent of his body surface burned and remained in the hospital a total of one hundred and forty-three days.[6] I was able to find 1 other case of recovery following extensive burns, a case reported by Bettman in which 80 per cent of the body surface was burned[7] Lam reported no cases of burns living if 75 per cent of the body was involved.[8] I am fully in accord with Lam[8] that it is "not foolish but wise to apply a good local dressing" when the patient is first seen. These burns were not debrided or cleaned except that the patient was placed under a shower of running water and no body surface was touched. No blisters were opened and only vaseline gauze was used with slight pressure dressings.

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SULFADIAZINE ANURIA, A LOWER NEPHRON NEPHROSIS

TREATMENT WITH INTRAVENOUS PROCAINE

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MONROE

Acute anuria, regardless of cause, presents the urologist with a most serious problem. Acute anuria secondary to pathological changes in the renal architecture, most commonly seen in the so-called lower nephron nephrosis syndrome, is one of the most difficult problems the urologist faces.

The present day routine treatment of lower nephron nephrosis is divided into three phases: (1) Treatment of shock if present. (2) Treatment of the anuria with special attention to a minimum fluid intake. (3) Treatment of the diuretic phase when established. These phases are common knowledge to the urological profession.

It is in the second phase of treatment that one tends to become discouraged and it is in this phase that suggestions are most readily sought and accepted. Early decapsulation is recommended quite often in the literature. From personal experience, one of us (ARB) has been very disappointed with the results in his use of that procedure. It is therefore with considerable enthusiasm that we call attention to an article by Friis[1] entitled "Sulfathiazole Anuria Cured by Means of Intravenous Procaine Treatment," and present a case of our own treated by intravenous procaine in addition to the present day accepted therapy. One case does not justify any positive statements, but certainly from our observation it does seem worthwhile that others try this method of therapy.

CASE REPORT

A 47 year old male, was treated with sulfadiazine by his family physician for an infection of his right arm. Prior to the arm infection the patient had been in excellent health. Following the administration of 18 grams of sulfadiazine the patient began to have some gross hematuria. There was no pain with the passage of the blood. With the onset of the hematuria oliguria appeared. The oliguria became progressively worse, and when we first saw the case on August 23, 1949, the patient was anuric. The NPN was 63, the creatinine was 7.15. Cystoscopy showed no evidence of ureteral blockage and no sulfadiazine crystals were noted in the washings. Vermooten[2] has called attention to the use of continuous gastric lavage. It is a very simple procedure requiring the use of tap water only for the lavaging medium. This type of lavage was instituted at the time of admission and served to keep the patient in relatively good condition throughout his period of anuria. Anuria had been present forty-eight hours and the NPN was 85 when 15 cc. of 1 per cent procaine were given intravenously on August 25, 1949. Immediately following the injection of the procaine the patient complained of pain over both kidneys and 50 cc. of bloody urine were passed through an indwelling urethral catheter during the next hour. No further urine was passed for the next twenty-four hours. At this time, on August 26, the procaine injection was repeated and during the next twenty-four hours the patient passed 100 cc. of urine. On the following two days, August 27 and 28, the patient passed 650 and 600 cc. of bloody urine, respectively. However since no urine was passed during the last twelve hours of August 28 or the first fifteen hours of August 29 and since diuresis seemed to have ceased, 15 cc. of 1 per cent procaine were again given intravenously about 3 P.M. on August 29. Diuresis immediately began and in the next twelve hours 925 cc. of urine were passed. By noon on August 30 urine was pouring from the catheter and on that day 5590 cc. of urine were passed. Then 4175 cc., 5300 cc., 5650 cc., 4420 cc., and 3279 cc. were passed respectively on the following five days and the patient began to recover rapidly. On September 6, the eighth day after full diuresis began, the patient was discharged from the hospital.

Throughout the entire course of the illness fluid intake was carefully regulated. Daily NPN, creatinine, CO_2 combining power, and serum chloride determinations were made. Gastric lavage was discontinued as soon as diuresis was in full force.

SUMMARY

The use of intravenous procaine in acute anuria is recommended for trial following

observations made on a case of sulfadiazine anuria.

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FRACTURES OF THE UPPER END OF THE FEMUR

AN ANALYSIS OF ONE THOUSAND CASES

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It has been stated that fractures of the upper end of the femur have the highest morbidity and mortality of any simple fracture of the long bones in middle and advanced age. The population is rapidly aging. Every year a greater proportion of the public is over 65 years of age than during the preceding year. Today there are 9,000,000 individuals over 65, and it is estimated that by 1980 this group will number over 22,000,000. Because fractures of the upper end of the femur occur in an older age group, the handling of these patients will constitute a major surgical problem.

Conservative treatment, which was formerly advocated for all cases of fracture of the upper end of the femur, has produced such poor results that more radical treatment is gradually replacing it. This is especially true of intracapsular fractures in which operative intervention has been followed by a far greater incidence of union and a lowering of the morbidity and mortality. While it is generally believed that intracapsular fractures are better treated by some form of internal fixation, there is some difference of opinion as to whether intertrochanteric and subtrochanteric fractures are best treated conservatively or radically. The development of surgical technics and improvements in anesthesia have made possible the salvaging of many patients who formerly would not have survived. Operability and mortality rates are improving, due, perhaps, to the use of antibiotics and chemotherapy in the prevention and treatment of pre and postoperative complications, and also, to a better understanding of surgical shock, blood volume, and thrombo-embolic complications.

The purpose of this report is to present a review of 1,000 cases of fracture of the upper end of the femur treated at the Charity Hospital of Louisiana in New Orleans. All of these cases were acute, fresh fractures; no pathologic fractures were included. The patients were admitted in rotation to four services. These statistics, therefore, represent the work, not of one surgeon, but of a representative group. An analysis and an evaluation of the methods of management exercised in these 1,000 cases may, therefore, yield information that will influence the selection of the future treatment of the patient who has sustained a fracture of the upper end of the femur.

AGE, RACE, SEX OF PATIENTS

The 1,000 patients studied ranged in age from 4 to 104 years. The average age of this group was 66.6 years. There were 522 white females, 306 white males, 102 colored females, and 70 colored males. There were 826 white and 172 colored patients, a ratio of approximately 7 to 1. The ratio of white females to males was 1.7 to 1, and the ratio of colored females to males was 1.45 to 1.

MORTALITY

There were 247 deaths, a mortality of 24.7 per cent. Of these 1,000 patients, 453 were treated by conservative measures and 143 died, a mortality of 31.5 per cent. The remaining 547 were treated by surgical means and 104 died, a mortality of 19 per cent. It is aparent that there is a significant difference between the mortality for those treated conservatively, 31.5 per cent, and for those treated by surgical intervention, 19 per cent. These figures will be broken down further for the different types of fractures of the upper end of the femur.

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TYPES OF FRACTURES

Fracture of the upper end of the femur may be classified as intracapsular, intertrochanteric and subtrochanteric. The intracapsular fractures may be further divided into subcapital, transcervical, and basilar, depending upon where, anatomically, the fracture line exists. The subcapital fracture is high on the neck and presents a problem in that it is quite difficult to get a good reduction and the incidence of nonunion is high. The transcervical fracture is through the middle of the neck and more distal than the subcapital fracture. The basal type is low on the neck near the trochanter but still within the capsule. Unfortunately, it was impossible to learn from the x-ray reports the exact anatomic location of the intracapsular fractures. These were reported as fractures through the neck or as intracapsular fractures as long as they appeared to be within the capsule of the hip joint. The x-ray department could be of great assistance by reporting accurately just where on the femoral neck the fracture line exists and the angle of the fracture, as these data are of considerable importance in the prognosis as to union.

INTRACAPSULAR FRACTURES

In this group of cases there were 504 intracapsular fractures. The youngest patient was 4 years of age, the oldest 104. The average age for this group was 66.3 years. There were 107 deaths, a mortality of 21.2 per cent.

Two hundred and seventy-eight patients were treated by conservative measures, including bed rest, manipulation, application of plaster casts, and some form of traction —Buck's extension, Russell traction, balanced skeletal traction, or Roger Anderson's well leg traction. There were 73 deaths, a mortality of 26.2 per cent. Only 12 patients could be followed for a period of a year or longer. In 43 per cent of these there was bony union.

Two hundred and twenty-six patients were treated by some form of internal fixation. The most common method of fixation was the use of the Smith-Peterson nail or double screws, all applied by the blind nailing method (the hip joint was not opened). There were 34 deaths, a mortality of 15 per cent. Of 78 patients followed for a period of a year or longer, 54 (69.8 per cent) showed bony union. Forty-seven of these surgically treated fractures had been fixed with Smith-Petersen nails and this technic produced 29 unions and 18 nonunions, a union rate of 61.7 per cent. The other 31 neck fractures were fixed with double screws. Of these 25 united and 6 showed evidence of nonunion, a union rate of 80.6 per cent.

Comment. There are many factors that influence union in this type of fracture. Damage to the superior and inferior capsular vessels, and vessels in the ligamentum teres, retards union just as accurate reduction and proper fixation with elimination of shearing stress promote it. Various types of nails, screws, pins, and bone grafts have been devised and employed to hold the fragments until union has taken place. Screws are mechanically more perfect for holding two fragments together than are the other means. They draw the proximal fragment to the distal fragment, are less traumatizing on insertion, and are relatively free from the danger of rotating or pushing the head away, which occasionally occurs when a large nail is driven. The proximal fragment, to unite, must be revascularized from the distal fragment and the bone of the proximal fragment must not be damaged if a good result is to be expected. The large Smith-Petersen nail cuts the neck and head into three compartments and occupies a large part of the neck and head. New formed blood vessels must grow around the nail. Screws or pins occupy less space and blood vessels may even grow in between them. Our experience with double screws in private practice fully confirms the evidence in this series, i. e., that the use of wood-type screws offers about 22 per cent greater chance for union.

INTERTROCHANTERIC FRACTURES

There were 438 intertrochanteric fractures. The average age of the patients in

this group was 70.3 years. There were 128 deaths, a mortality of 29.2 per cent.

Two hundred and sixty-three were treated by some form of internal fixation. In this group there were 71 deaths, a mortality of 26.9 per cent.

One hundred and seventy-five were treated by conservative measures, most commonly Russell and the well leg traction. There were 57 deaths, a mortality of 32.2 per cent.

Eighty of the patients were followed for a period of six months or longer. There were no instances of nonunion.

Comment. Because intertrochanteric fractures are extracapsular and nonunion rarely results, there are many who believe that this fracture should be treated by conservative measures. Yet the mortality rate was slightly lower, 26.9 per cent, in those who were treated by internal fixation than in those treated conservatively, 32.5 per cent. The difference is not great, but it does show a trend towards a more successful outcome.

The fact that the lowering of the mortality by surgical intervention is not so great as in the intracapsular group can be explained by several factors.

1. If internal fixation is used, the operation is more extensive than that used in fractures of the neck of the femur, and naturally, a higher mortality can be expected.

2. One of the chief difficulties in the application of internal fixation devices in intertrochanteric fractures is that the fracture line is often comminuted. X-ray studies do not always show this and comminution is only found when the fragments are exposed.

3. In our series, the average age of the patients with intracapsular fractures was 66.3, and the older age carries a greater risk.

4. All of the patients in the present series were charity patients with extremely low means. Practically all in this age group were undernourished and many of them had been or were being treated for chronic medical conditions. In a smaller number of privately treated patients, the mortality was 12 per cent. Funsten and Frankel report a group of 49 cases treated by surgical intervention, with a mortality of only 8 per cent. Again the average hospital stay for this group of patients was 55 days. In some series, patients are allowed to go home on or about the fifteenth day, and should they die at home at some time thereafter, they do not appear in the hospital mortality rate.

Intertrochanteric fractures are an entirely different problem in the young. Since nonunion is practically unknown, any method that will reduce the fracture and maintain it will give good results.

The mortality in this "poor risk" group is admittedly far too high. However, despite the factors enumerated, there is a reduction in the mortality, and there are still other advantages that must be taken into consideration, such as immediate relief from pain, freedom from confinement, and easier maintenance of the general physical condition of the patient.

SUBTROCHANTERIC FRACTURES

There were 51 subtrochanteric fractures in this group. The average age was 59.9 years. There were 12 deaths, a mortality of 23.5 per cent. Eleven of the 51 patients were treated by internal fixation and 2 died, a mortality of 18.1 per cent. Of 40 patients treated by conservative measures, 10 died, a mortality of 25 per cent. Twenty-eight patients were followed for a period of six months or longer. There were no nonunions.

Comment. (1.) This fracture occurs in a younger age group than the intracapsular and intratrochanteric types. (2.) When displacement does occur, and it usually does, reduction is often difficult and is rarely obtained by closed methods. (3.) The short proximal fragment is abducted by the pull of the strong gluteal muscles. The adductors, which are inserted at a lower level, pull the distal fragment medially. If the fracture line lies below the lesser trochanter, the proximal fragment is also flexed by the iliopsoas. The short rotatores externally rotate the proximal fragments.

(4.) Nonunion is no problem if the fracture can be reduced and maintained. (5). This can best be accomplished by open reduction which is also accompanied by a lower morbidity and mortality.

COMPLICATIONS

Of the 1,000 patients, 341, or 34.1 per cent, developed decubitus ulcers. These varied from small superficial erosions of the skin to large deep sloughs that extended to the sacrum. In diameter they varied from 2 to 20 cm. with considerable undermining. Some patients developed them on the first day in bed. Seventy patients had urinary incontinence, 4 fecal incontinence, and 24 were listed as having both. It is surprising that no deaths in the conservatively or surgically treated group were directly attributed to pulmonary emboli. Furthermore, none of these patients received anticoagulant therapy or had femoral vein ligation. In 260 cases hypertensive heart disease was present in a degree severe enough to make the outcome doubtful. Other complications listed in order of frequency were pneumonia, senile dementia, uremia, postoperative psychoses, cerebral vascular accidents, and diabetes mellitus. It is apparent, therefore, that the presence of severe complicating diseases and their nature exert a significant and often decisive influence on the survival of the patient. Pneumonia or heart disease, or a combination of both, accounted for 89 per cent of the deaths. Pre-existing ailments and complications not directly attributed to the operation were the greatest factor in the mortality rate. Co-operation with the internists and other specialists is essential.

One of the most distressing complications was the loss of the will to live. Many of these patients were alone in the world. They felt that they had outlived their usefulness and were a burden to their children and relatives. Having nothing to look forward to they lapsed into a state of indifference and pursued a gradual downhill course to death. A vigorous and active social service and rehabilitation program would obviously be of great help.

ANESTHESIA

Local or a light general anesthesia was the most common anesthetic agent used. Local 1 per cent novocaine alone was used in approximately 30 per cent of the cases. Local 1 per cent novocaine and general anesthesia, with either nitrous oxide or ethylene, were administered in 60 per cent; and general anesthesia alone, either nitrous oxide or ethylene with ether, in 10 per cent.

It is very difficult, and even impossible, to draw any conclusions regarding the effect of any of the anesthetic agents used in this series on mortality as there were no deaths on the operating table or immediately after operation. While pneumonia was listed as causing 42 per cent of the deaths, it could not be definitely stated that the anesthetic was a contributing factor as most of the deaths occurred 10 to 15 days after the operation.

Comment. With the skilfull use of the anesthetics now available, the anesthetic has not contributed a grave risk and has had little or no effect on the mortality.

AVERAGE HOSPITAL STAY

The average time spent in the hospital for a fracture of the upper end of the femur was 55 days. Those patients treated by conservative measures averaged 73.3 days, while those treated by internal fixation remained 42.5 days. The type of fracture did not seem to influence the time to any significant degree. The time for the surgically treated group seems unnecessarily long when compared with the hospital stay of a group treated in a private institution, which averaged 15 days. This can partially be explained by the fact that expense was no item. It was "on the house" and frequently the patients' relatives were in no hurry to take them home.

DEATH IN DECADES

There were no deaths below the age of 40, only 1 between 40 and 50, and 5 between the ages of 50 and 60. The majority of deaths occurred between the ages of 60 and 90, and 70 per cent occurred between the ages of 70 and 90. There were 160 deaths in females and 96 in males.

FOLLOW-UP

It is the practice at Charity Hospital of

Louisiana to follow these patients at regular intervals in the outpatient clinics. We were quite disappointed that our follow-up group was so small, and that the majority of the patients followed were observed for less than a year. This was due to various factors such as age, disability, distance from the clinic, transportation difficulties, and lack of funds. Nevertheless, we feel that the results obtained are significant and some conclusions may be drawn from the series.

CONCLUSIONS

1. Intracapsular fractures when treated by operative means were followed by a higher rate of union as well as a lowered mortality. There was a 22.2 per cent higher rate of union when screws were used in preference to the Smith-Petersen nail.

2. Intertrochanteric fractures are a different problem under the age of 45, and may be treated either conservatively or by internal fixation with good results. Over 45 there is a gradual rise in the mortality, whether treated by surgical intervention or conservatively, and slightly better results are obtained with internal fixation.

3. Subtrochanteric fractures occur in a lower age group than the other types. When internal fixation is used it is accomplished by a lowered morbidity and mortality.

4. Anesthesia in the operative group did not contribute a grave risk and had little or no effect on the mortality.

5. The average number of days spent in the hospital was 73.3 for the conservatively treated group, 42.5 for those treated by surgical procedures.

6. The morbidity, mortality, and the incidence of nonunion in all types of fractures of the upper end of the femur have been reduced by internal fixation.

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BETA IRRADIATION IN OPHTHALMOLOGY*

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It is both necessary and appropriate, before discussing the clinical possibilities of beta irradiation in ophthalmology, to outline briefly certain fundamental considerations concerning this method of treatment.

BETA IRRADIATION

Properties of Beta Rays. Radium emanations consist of alpha, beta, and gamma rays. Alpha rays have no power of penetration, and therefore, have no therapeutic usefulness because irradiation, naturally, is without effect unless it reaches the tissues to be treated and is absorbed by them. Gamma rays, which are hard rays of high penetrating power, are used in the ordinary forms of radium and roentgen ray treatment. Beta rays, which are negatively charged electrons, can penetrate no more than 10 mm. at most and their passage can easily be stopped by appropriate screens of

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platinum, brass, lead, silver, or other metals. When these rays are used therapeutically, practically all of their effect disappears within the first 3 mm. of tissue which they traverse and 75 per cent is absorbed within the first 2 mm.

The surface absorption of beta rays, as it might be termed, makes them peculiarly suitable for the treatment of lesions of the external eye. Furthermore, since all radiotherapy is predicated on the differential sensitivity of diseased tissue, which is to be destroyed, and normal tissue, which is to be protected, the structure of the external eye and the disease processes which may occur in it make this region peculiarly suitable for this form of treatment. According to Desjardins and Williams,[1] tissues can be listed in the order of diminishing radiosensitivity as lymphoid cells, polymorphonuclear leukocytes, epithelium, endothelium, connective tissue, muscle, bone and nerve.

Method of Application. Iliff, Burnam and Neill, Ruedemann, and others with access to radon plants have employed radon in their work with beta rays and calculate the dosage in gram seconds. In the absence of these facilities we have done our work with radium and have continued, as a matter of convenience, to express our dosages in the usual terms of grams and minutes.

The intensity of irradiation varies inversely with the square of the distance of the applicator from the target, which means that a slight variation in distance can result in a large difference in the effective dosage. For this reason the spray technic (in which the applicator is moved slightly back and forth 1-3 mm. above the tissue to be treated) gives an uneven distribution over an area which itself is not clearly outlined. Hughes and Iliff[2], who formerly used the spray method, have now discarded it and give all their treatments by direct contact between the lesion and the applicator.

The Burnam applicator is suitable only for the application of radon. Our own work has been done with the Iliff applicator, which contains 50 mg. of radium and which permits contact over a surface 10 mm. long and 5 mm. wide. The monel metal, of which the applicator is constructed, is 0.1 mm. thick and the handle is 12 inches long.

Neither the Burnam nor the Iliff applicator can correctly be termed a beta ray applicator because both permit the passage of gamma as well as of beta rays. Burnam and Neill[3] consider the term justified, however, because gamma rays do not pass through the filter in sufficient amounts to cause injury to the deeper structures of the eye and because the effects produced have been shown to be the result of absorption of beta rays.

The applicator devised by Swanberg[4] for the delivery of radium "D" furnishes a treatment surface 9 mm. wide and a radioactive diameter of 5.6 mm. Theoretically, pure beta irradiation can be obtained by utilization of this agent, which is a member of the radium uranium series and is a disintegration product of radium element. Since it produces beta rays intensively but gives off only a few gamma rays, it is theoretically ideal for the treatment of lesions on the cornea, the normal thickness of which is about 1 mm. Another desirable property of radium "D," which is still available only in limited quantities, is that a relatively small amount can do the work of a much larger amount of radon and radium element. Unlike radon, which depreciates at the rate of 50 per cent every four days, radium "D" is relatively stable and depreciates at the rate of only 2.25 per cent per year, a reduction of 50 per cent thus not occurring for more than twenty-two years.

When 10 mc. of radium "D" is incorporated into the Swanberg applicator, the amount of radioactive material per square millimeter of surface area is eight times that of the full-strength radium element plaque. Moreover, the effective beta ray output is additionally increased by the fact that the treatment surface has an aluminum filter 0.05 mm. thick, the density of

which is about a sixth of that of the monel metal filter used in the Iliff applicator.

Highly successful results, according to Swanberg, were obtained by Ruedemann and others who used this applicator at the radium "D" clinic established at the City of Detroit Receiving Hospital, and only recently I have seen the excellent results obtained with it by Okrainetz at the Manhattan Eye and Ear Infirmary. Our own results with it have been extremely disappointing, though we found, as we had suspected, that the instrument which we were using was defective. These defects are now being rectified, and we look forward to securing satisfactory results with it when it is returned to us.

Beta irradiation must be applied with the utmost care from the standpoint of both patient and physician. Since the skin erythema dose of unfiltered beta rays is 18 gm. seconds, while that of gamma rays is 8 gm. seconds, adequate beta irradiation can be applied with such brief exposures that the effect of the gamma irradiation delivered simultaneously may be largely discounted. The treatment time must be determined accurately, with a stop watch, and very careful records must be kept. The duration of treatment must be strictly limited. We have not at any time exceeded ten minutes per application, nor have we ever treated a patient oftener than once a week. The applicator is handled with caution and the direct opening is applied only toward the lesion to be treated, never toward any other part of the patient's body or toward the ophthalmologist.

From the standpoint of the ophthalmologist, a daily tolerance dose of 0.1 r must not be exceeded. By a series of studies with the Geiger counter we have learned that this limit is reached in five minutes when the distance of radium from operator is 6 inches, but that, when the distance is 18 inches, exposure for ten minutes is possible before the limit of safety is reached.

While we usually limit our personnel to the treatment of one patient each day, we have found it possible to treat more than one case within twenty-four hours by the use of a plastic contact lens, in which a window has been cut the exact size of the applicator. After the eye is anesthetized with pontocaine the lens is applied, with the window over the lesion to be treated. The applicator is then put into position and the patient can be left unattended for the duration of the treatment.

Reactions. No injuries of the lens, cornea or other structures seem to have been reported as the result of the absorption of gamma rays in the course of beta irradiation. The possible immediate reactions from beta irradiation are chiefly conjunctivitis with congestion, edema, and a serous or mucopurulent discharge. Later damage includes scarring, adhesions to the globe and lids, and ectropion. Damage to the iris is manifested by pain, swelling, and the formation of posterior synechiae. Corneal damage usually takes the form of ulceration, which may go on to perforation. Ruedemann[5] mentions "several" cases in which excessive treatment of severe corneal leukomas resulted in small perforating ulcers, for which enucleation was required.

Cataract formation is a delayed reaction, which may come on two years or more after exposure. We have seen no instance of such reaction in patients whom we have had under observation between two and three years, and expect none, because of the extreme caution with which we have used this method. Ruedemann[5], and Hughes and Iliff[2], have had a similar experience. That cataract formation can occur, however, is evident from our experimental studies on rabbits[6], in practically all of which cataractous changes were noted, although they might have been expected, because of the large dosages deliberately used. Our own reactions have been limited to an occasional instance of hyperemia, which was always transitory, and it seems fair to say that the minimal reactions generally reported in the literature are evidence of the caution with which this method seems to have been used.

Beta irradiation apparently has no stimulating effects. In fact, as Hughes and Iliff[2] emphasize, all the evidence is to the

effect that regenerative processes are retarded.

CLINICAL USAGES

To date, beta irradiation has been employed in a wide variety of ophthalmic diseases and conditions. It has been most effective (1) in small, superficial growths, such as epithelioma, angioma, papilloma, carcinoma, lymphosarcoma, pigmented mole, melano-epithelioma and postoperative keloids; (2) in certain infections, such as scleritis, particularly of the tuberculous variety, keratitis, and resistant conjunctivitis; (3) in vernal conjunctivitis; and (4) as a preoperative and postoperative measure in keratoplasty and keratectomy. Woods and Burch[7] employed beta irradiation after iris inclusion operations in negroes, on the ground that the tendency of this race is to form excessive amounts of scar tissue, which would be likely to have a compromising effect on the filtering scar. Their results in a small series of cases were disappointing. Fridenwald[8], however, reported excellent results when he used this method in white subjects, after corneal scleral trephine.

As Hughes and Iliff[2] point out, the results of treatment secured by various workers with beta irradiation are difficult to assess comparatively because of differences in dosage and technic, incomplete descriptions, varying distances of application, possible cumulative effects, and ignorance of the differential sensitivity of normal and pathologic tissue. Moreover, as they also note, there has been an unfortunate tendency to use the beta irradiation clinic as a convenient spot to which hopeless cases can be referred for treatment, often without either theoretical or practical justification.

Our own clinical experience with beta irradiation, which now covers a period of three years, has been limited to vernal conjunctivitis, certain superficial growths, occasional infections, and glaucoma, in addition to its use as a preoperative and postoperative measure.

Vernal Conjunctivitis. Excellent results have been reported by all workers with beta irradiation in both the palpebral and the limbal form of vernal conjunctivitis, especially in the early cases, in which the lymphoid cells, young fibroblasts and blood vessels are all highly radiosensitive. Results are naturally less prompt in the older cases of the so-called pavement-stone type, which contain radioresistant areas of hyaline degeneration and old fibrous tissue.

Our own experience consists of about 15 cases, in which, according to the severity of the manifestations, the number of applications ranged from one to five or six, given at weekly intervals, for two to three minutes per treatment. Without exception, burning and itching were relieved promptly and in the palpebral variety it was possible to observe gross shrinkage in the size of the follicles. Beta irradiation is apparently the best treatment now available for vernal conjunctivitis. It is certainly far superior to the methods formerly employed, none of which accomplished more than transient improvement. Since this is a seasonal disease, it might be expected that treatment would have to be repeated yearly, but only 2 of the 15 patients whom we treated in 1947 or 1948 are finding it necessary to be treated again this year.

So far we have not seen any case in which it seemed necessary to employ Ruedemann's[5] plan of excising large and fibrotic polyps before beta irradiation was applied. For milder lesions he uses the spray method primarily. He has had particularly good results with beta irradiation in vernal conjunctivitis in childhood.

New Growths. The general principle of beta irradiation in new growths is to limit its primary employment to growths which are small, shallow, and superficial, and which would be difficult to excise or to treat with ordinary methods of irradiation. Beta irradiation is not a satisfactory form of treatment, at least primarily, for growths of any depth, though it may be used as a secondary measure after surgical excision.

To date we have used beta irradiation in

2 superficial carcinomas, with good results in both. We have also employed it in 7 papillomas, usually for a total of fifteen minutes in three weekly applications. All of these growths were located on the lids, the conjunctiva, or the limbus. We have used it in 6 angiomas, the cases being selected and the amount of treatment applied according to the age of the blood vessels. If the vessels were newly formed, treatment for a maximum of eight minutes gave excellent results. When the vessels were larger and older, treatment for as long as twenty to twenty-five minutes was necessary, in five minute amounts at weekly intervals. We have not attempted to use this method in the port-wine type of angioma, in which good results could scarcely be expected.

Over the past two years we have treated 25 pterygia with beta irradiation, which usually was the primary form of treatment. All of these cases were carefully selected. Small growths were treated for ten to fifteen minutes in two or three treatments. Large growths were submitted to surgery and beta irradiation was used as a postoperative measure if any tendency to recurrence was apparent. To date there has been no recurrence in any of the 40 new growths treated by beta irradiation.

Infections. The use of beta irradiation in infections rests upon rather uncertain grounds. The method is frequently effective, but why it should be is not clear. As Pendergrass and Hodes[9] have shown, the dosages have been shown to depress the defense mechanism, especially the reticuloendothelial system. Hughes and Iliff[2] list as possible reasons for the good results: (1) destruction of leukocytes with liberation of proteolytic enzymes and antibodies; (2) liberation of antibodies from other sources; and (3) production of active hyperemia. The successes achieved in such chronic infectious granulomas as tuberculosis can be attributed to the radiosensitivity of epitheloid and giant cells, which are replaced by fibrous tissue, and possibly to the liberation of antibodies.

How successful beta irradiation is in anterior ocular tuberculosis is debatable. Although Woods[10] and Iliff[11] reported 72 cases, in all but 8.3 per cent of which healing or improvement occurred, and in all but 12 per cent of which vision was improved or was maintained at a stationary level, they frankly admit that the wide variation in the characteristics and ultimate prognosis of this type of ocular tuberculosis makes their statistics of doubtful worth.

We have not used beta irradiation for anterior ocular tuberculosis because we can see no rationale for its use. On the other hand, we have under observation at this time 2 patients with tuberculous episcleritis who were given beta irradiation for twelve minutes each about a year ago and both of whom have since been free from exacerbations and recurrences.

We have also treated by this method 6 cases of interstitial keratitis of syphilitic origin, with the objective of controlling the vascularity of the cornea. Routine antisyphilitic treatment was employed at the same time. Except for this particular manifestation, we can see no justification for the treatment of ocular syphilis by beta irradiation, nor do we see the rationale of employing it for such constitutional infections as brucellosis, even when there are ocular manifestations.

Preoperative and Postoperative Usages. Beta irradiation has been used before and after keratoplasty and keratectomy, to prevent the development of corneal vascularization, or to control it if it has developed. We have employed it in this manner in 10 keratoplasties and in 8 keratectomies, and we find it interesting to recall that when we first began to employ these operations, some ten years ago, we used radium for this purpose, applying it by the conventional method and getting very good results.

Since a heavily vascularized cornea militates against a successful result in corneal transplantation and in keratectomy, it seems quite rational to apply beta irradiation for about twenty minutes, over a four week period, at least eight weeks before

operation, or to apply it after operation if vascularity becomes at all prominent.

The dosage of beta rays employed depends upon the degree of vascularization, and careful selection of cases is necessary. Repeated small doses, as Hughes and Iliff[2] emphasize, are better than single large doses, because the cells are caught in the sensitive, premitotic stage, and rapidly proliferating endothelium of capillaries growing into the cornea can thus be inhibited. This method of application, furthermore, is a protection for normal sensitive tissue. If the vessels are numerous and old, the results of beta irradiation are less good. In fact, as Hughes and Iliff[2] showed, the fibroblasts in such scars are sensitive to irradiation but fibrocytes are not. Furthermore, dosages of beta irradiation large enough to destroy old fibrous tissue are unlikely to have anything but a detrimental effect, in that normal stromal cells would thereby be destroyed and normal regenerative processes would be retarded. Finally, irradiation during the stage of active proliferation of fibroblasts may diminish the ultimate amount of scarring, but possibly only at the risk of perforation of the newly damaged cornea.

All of these objections are well taken. It must also be admitted that, especially in children, increasing clarity of the cornea may occur over a period of years, together with a reduction in the size and intensity of opacification, in the absence of any treatment at all. On the other hand, if the method is used carefully, our personal experience suggests that it is of value. Perhaps our results would have been equally good if we had omitted beta irradiation, but is certainly seems to have done no harm.

Glaucoma. Our most interesting experiences with beta irradiation have been in glaucoma, for which we have employed it with the objective of decreasing the production of intraocular fluid at the source, by destruction of the ciliary body. All other therapeutic methods, except cyclodiathermy, attempt to increase the rate of flow of the intraocular fluid through normal drainage channels. Cyclodiathermic methods have achieved reductions of intraocular tension, but they have invariably been short-lived.

Our work in glaucoma began with a series of experimental studies on rabbits, by which we were able to demonstrate that beta irradiation could produce the desired vascular changes in the endothelium of the ciliary blood supply. Clinical testing has been carried out very cautiously, and to date we have used the method in only 9 cases, because we have not employed it in any instance in which standard methods had not been given a full trial or in which it seemed that these methods could achieve nothing. These 9 cases include:

Six cases in which glaucoma developed after cataract operations. All the patients had been treated by standard measures, including cyclodialysis, iridotasis, or iridectomy.

One instance of untreated primary glaucoma, in which the intraocular tension was 90 (Schiotz) when the patient was first seen.

One instance of primary glaucoma, for which one eye had already been removed. Two filtration operations had been performed, without effect, on the remaining eye.

One instance of congenital buphthalmos in a child who was four years old when he was first seen, in November, 1946. Staged iridotasis was performed, the operation on the right eye being followed by massive prolapse of the iris and ciliary body, with intraocular hemorrhage, for which enucleation was finally necessary. Tension remained high in the eye still in situ, in spite of adequate therapy, and almost in desperation beta irradiation was finally resorted to at the end of ten months. It should be noted that in this case, as in all but 2 of the other cases, the lens had already been removed and that the risk of cataract formation therefore did not exist.

One speaks of results with hesitancy when the period of observation is so short.

Every patient treated by beta irradiation has, however, shown improvement. Exclusive of the child just mentioned, who has been observed for nineteen months since the first treatment and for sixteen months since the last, the period of observation has been sixteen months in 1 case, eleven months in 4, and seven to eight months in the 3 remaining cases. In every instance there was a drop, usually marked, after the application of beta rays, and in only 2 instances did the tension ever again exceed the tension present when treatment was instituted. At the present time every patient has a lower tension than when treatment was begun, and in 2 cases in which the initial readings were 30 and 40, respectively, the latest readings are 12 to 20 (Schiotz) respectively.

As to the child with congenital glaucoma, his condition was definitely progressive before beta irradiation, but it has not progressed since, and there has been no further loss of the (very limited) vision he possessed when he was first seen. Moreover, the consistently lower intraocular tension suggests that the disease process, the natural tendency of which is progressive, has become at least stationary if it has not actually regressed. The relatively long duration of the improvement—now nineteen months—is the most hopeful feature of the case and is in contrast to the disappointingly brief decreases in intraocular tension reported by Troncoso[12] as the result of diathermic therapy.

SUMMARY AND CONCLUSIONS

Beta irradiation has now been established as a satisfactory method of treatment for certain diseases and conditions affecting the internal eye, including vernal conjunctivitis, superficial new growths, and certain infections. It is useful as a preoperative and postoperative measure in keratoplasty and keratectomy. Finally, it seems to offer some promise in glaucoma, by reducing intraocular pressure through control of aqueous production at its source in the ciliary body. The background of beta irradiation has been briefly outlined, methods of application have been discussed, with emphasis on the precautious necessary, and the results secured in a personal experience with the conditions mentioned have been reported.

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DISCUSSION

Dr. Wm. B. Clark (New Orleans): Dr. Haik is to be congratulated on his excellent presentation of a very timely subject. There seems to be much confusion and misapprehension about the use of this excellent source of potent radiant energy and its aid in treating lesions around the anterior segment of the eye. I am sure that one who reads Dr. Haik's paper carefully will see that most of these misconceptions are not justified. Sufficient experimental work, supplemented by clinical experience, has been done to prove that the use of beta irradiation in ophthalmology is a safe procedure if used in the manner recommended by those who have had the most experience with it.

My own experience with its use dates back to the purchase of the so-called Iliff beta radium applicator by the New Orleans Eye, Ear, Nose and Throat Hospital on April 2, 1947. This instrument is available on a rental basis to all ophthalmologists who wish to use it. Since that time we have treated 64 eyes with the applicator with very gratifying results in most cases, and disappointments in others.

When the Swanberg applicator, mentioned by Dr. Haik, containing 10 millicuries of radium-D became available in January, 1948, we purchased one of these applicators at the Smith-Clark Eye Clinic. Unlike Dr. Haik, however, our results with this

instrument were very satisfactory. There is only one objection: In order to get the sufficient amount of dosage from the instrument it is sometimes necessary to hold the applicator in contact with a lesion for as long as ten to fifteen minutes. This, of course, is very tiring to both the patient and the operator. Several mechanical devices have been designed to hold the applicator in contact with a lesion on the anterior segment of the eye. In my experience, none of these are satisfactory for some patients are unable to hold their eye in one position of gaze and the applicator is displaced from its proper position in contact with the lesion. There is nothing, so far as I know, that will serve as a substitute for standing by the patient with the applicator held manually in contact with the lesion.

In order to overcome this objection to the original Swanberg radium-D applicator, we now have on order one of the more recent models as designed by Dr. A. D. Ruedemann of Wayne University. This applicator is of similar design but contains twice as much, that is, 20 millicuries, of radium-D. This applicator is made by painting the radium-D on a small sheet of monel metal, in successive layers. Each layer is allowed to dry before the next is placed on. With this layered arrangement some of the beta rays from the deeper layers are absorbed by the superficial layers. It has been estimated that the loss in efficiency varies from 10 to 25 per cent. Thus, it is obvious that with the 20 millicurie applicator one will not get twice the effect as with the 10 millicurie applicator. Therefore, in figuring the dose of the larger applicator, this must be taken into consideration.

In our opinion the Swanberg radium applicator has a distinct advantage in that the patient and the operator are not exposed to gamma radiation. Thus, a series of one or more patients can be treated in one day without danger to the operator.

Our clinical results have been most satisfactory in the following conditions: (1) Vernal conjunctivitis, both the limbal and palpebral types; (2) recent corneal scars; (3) superficial early corneal vascularization; (4) episcleritis; and (5) regeneration of blood vessels in the cornea where keratoplasties have been done.

While one does not have the opportunity to treat large numbers of cases of tuberculous anterior uveitis, it was our good fortune to have a patient who was one of the series reported by Dr. Alan Woods of Johns Hopkins Hospital and referred to by Dr. Haik. The results in this case were so spectacular that I have used beta irradiation in 2 of my own cases. These results were equally gratifying.

Our results in destroying large interstitial vessels in the cornea have been rather disappointing. Of course this can be understood when one realizes how beta irradiation influences tissue, as Dr. Haik has so ably explained.

Our most spectacular results have been in cases of early corneal scars. However, a word of caution should be expressed here about the use of this form of therapy in the treatment of corneal lesions such as chemical burns in which one half or more of the corneal stroma has been damaged. I recall one case in particular of a rather severe lime burn with early pannus, which was seen late. This was treated with possibly a little too much enthusiasm, and while we ended up with a fairly clear cornea, the remaining stroma was so thin the patient developed a keratoconus. This will ultimately have to be repaired with a perforating keratoplasty, a procedure which could probably have been done more successfully in an opaque cornea rather than in a conical cornea.

Our experience with the use of beta irradiation in glaucoma has been limited to a small series of cases of aphakic glaucomas in which our results have been disappointing.

Dr. Green: I may be presumptuous in saying this, but speaking from a radiologist's standpoint, I want to again caution you about using radium. By that, I don't want you to quit using radium, I think it is a very valuable therapeutic agent. I hope that you realize the dangers of using radium with your bare hands, in any type of applicator that is not thick enough to keep out all of the radiations that may be thrown to you or to your hands. It is pretty tragic for a surgeon, as a surgeon, if he loses a finger, but handling large amounts of radium with the bare hands is dangerous and should not be used every day. I know that the applicator is away from your hands, but you are going to find that you are going to get body exposure, and your hands are closer to it, and you get more exposure to your hands and for a longer period of time.

Any type of radiation, whether alpha, beta or gamma can be injurious to the hands; and when they are once injured, the action is not reversible. As you become older, the hands are more abused, and there is greater possibility of tumor from the radium.

I just want to make that warning, but I hope it is not a warning that will discourage anyone from the use of the beta irradiation in treatment of the eye.

Dr. Louis A. Breffeilh (New Orleans): Although much has been written concerning the use of radium or radon in ophthalmology, very little has been published concerning the danger to the operator and patient. What is the tissue reaction? What is the best dosage and what dosage may be tolerated by the operator are a few unanswered questions.

Much has been written on the rabbit experiments in which lens changes were not present but most of this work was conducted with radon in heavy dosages and not with the applicators now on the market. Our first experiments revealed that over

thirty minutes dosage in rabbits produced lenticular changes. The applicator used contained 50 mg. of radium and is the one on the market.

Fortunately, none of the humans examined and treated have revealed any evidence of lenticular changes. We will continue our cautious study for these changes.

The next problem is the safety of the operator. This problem concerns us as doctors and I fear if we wait for the producers of the radium, then a satisfactory answer will not be obtained. A minometer indicator was used to determine the safe dosage and the work was performed at the New Orleans Charity Hospital. At first the indicator was attached to the wrist of the operator and another at his belt. The applicator was held on the tip of its handle and after twenty minutes the wrist indicator revealed the dosage unsafe, while thirty minutes was needed for the belt indicator to pass the safety exposure. Since the dosage is inversely proportional to the distance the experiment was then conducted with the indicator held first 6 inches from the tip alongside the handle. The indicator revealed the safe dosage was under five minutes. At the distance of 18 inches, the indicator revealed safe dosage under ten minutes.

It is almost impossible for the operator to hold the applicator in place for five minutes at the tip of the handle, consequently the elbow is usually rested near the patient within the 6 inch distance. From the above study it was necessary to devise a method of holding the radium in place if more than one patient is to be treated a day, thus the contact lens holder was devised.

Dr. Haik (In conclusion): I would like to thank Dr. Clark, Dr. Green and Dr. Breffeilh for their discussion of this paper. I had asked Dr. Green if he would not discuss the dangers in the use of radium in ophthalmology. I thought it was important for us to know the dangers and how we could protect ourselves. We get very little information out of the literature and very little from experimental reports on the care of the individual handling the radium.

Dr. Clark mentions the use of beta irradiation in intraocular tuberculosis and in our paper I refer to the work of Woods and Iliff and their results.

Dr. Green: When you have this applicator in your office, the company from which you buy it, do they send you a way to store it so that you are not exposed to it?

Dr. Haik: We have a three-inch lead compartment in which to keep our radium and this is the one recommended by the American Radium Society. It is kept in a separate room and there is little or no exposure to anyone in the office.

Dr. F. E. Le Jeune (New Orleans): We were concerned in the use of radium in otorhinolaryngology, and were told by the company from which we obtained the applicator that the three inch lead was not sufficient to protect those who came in contact or entered the room where this radium was stored. Eventually we were made to get a six-inch lead container. That is what we are using at the present time. We only use the three-inch one to transport the applicator to the hospital or the various rooms; otherwise it is kept at all times in the six-inch lead container.

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SURVEY OF PHYSICIANS' INCOMES

In fighting the battle against State Medicine and in improving the political relations of organized medicine, knowledge of physicians' incomes has become of great importance. We need to know just what the public pays doctors.

Surveys of physicans' incomes have been conducted in the past, and one as recent as 1941. Investigators in this field consider that in the past ten years changes have occurred, and they also have reason to believe the averages in the past have been too high. It is felt that this error came into the figures for the simple reason that those physicians who did not have bookkeepers could not find figures readily available and did not fill out the questionnaires sent them. Accordingly, it is urged that every physician who receives a questionnaire complete it, no matter how small the income. It is only by this means that a representative amount of data can be compiled. With this data, a complete picture can be presented to the public.

Within the next month, the Bureau of Medical Economic Research of the American Medical Association, and the Office of Business Economics of the U. S. Department of Commerce will jointly conduct the survey. The A.M.A. participation has been authorized by the A.M.A. Board of Trustees. The results of the survey will be published by the Department of Commerce next fall in its monthly publication, "Survey of Current Business." Surveys of incomes of dentists and lawyers have already been made jointly with the American Dental Association and the American Bar Association, within the past year. The analyses were published in August 1949, and in January 1950, respectively.

The physician is assured that the survey has no relation whatever to the operations of the U. S. Bureau of Internal Revenue. It is stated that there is no way by which the Department of Commerce could have obtained the needed information from the Bureau of Internal Revenue. Considering the way that various Government bureaus and departments fight among themselves, this is quite easy to believe. By the participation of organized medicine in the survey we have added assurance of its correct interpretation.

The procedure to be followed is one which will assure complete impartiality. The Bureau of Medical Economic Research of the A.M.A. has files of about 200,000 physicians. The survey will cover 125,000 or 62½ per cent of the total. There will be a short form for income data for the year 1949 only. There will be a long form questionnaire for the years 1945 through 1949. All are to be returned unsigned in franked envelopes. The short form will be sent once only to every other name in the file. Of the remaining 100,000 names, every

fourth will be selected. Those who comprise this fourth will receive 10,000 short forms and 15,000 long forms. These 25,000, who may receive either the short or the long forms, are requested to return the forms in a franked envelope which will carry a code number. This code number will identify the physician to the Bureau of Medical Economic Research. It does not appear on the questionnaire and will not identify it in any respect. The only purpose of the code number is to enable the Bureau of Medical Economic Research to address a follow-up letter to those not replying to the first request.

Physicians will be doing the medical profession a service by filling out the forms and returning them just as soon as they receive them.

Statistics which will be accumulated in a complete survey, such as we hope this will be, will give factual support to an observation which is all too well understood within the profession and too little appreciated outside of it. Considering the length and expense of preparation, the hours of work, and the limited years of productivity, the average physician is underpaid. This survey will also show more conclusively than those in the past that the major expense in illness is not physicians' services.

Each physician, therefore, who receives a questionnaire is urged to fill it out completely and promptly.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

DR. EDWIN HUGH LAWSON

As the Seventieth Annual Meeting of the Louisiana State Medical Society comes to an end this year, Dr. Hugh Lawson will conclude his term of office as President. The State Society pays tribute to his direction of the executive office during this year. Owing to the fact that problems have been numerous and complex, the year has been an arduous one. Matters of scientific moment have claimed the attention of the President and of the Executive Committee more this year than in others past. In addition, the threat of State Medicine and the impact of the agitation to destroy private practice made it necessary for the President to direct the organization's activities to combat these influences.

Dr. Lawson has served the Society's interests exceedingly well, and we can count ourselves fortunate that in times as troublesome as these we have had such splendid leadership. It is, however, by no chance circumstance that Dr. Lawson was able to guide the affairs of the Society and serve so admirably its aims and purposes.

Dr. Lawson was born in Indianapolis, Indiana, on May 13, 1889, the son of the Reverend M. M. Lawson and Katherine Darrah.

He attended grammar and high school in Nashville, Tennessee, and the University of Arkansas at Fayetteville from 1916 to 1920, graduating with a Bachelor of Science in Chemistry. He entered Tulane University Medical School in 1920, and was graduated in 1924, with the degree of Doctor in Medicine. This began a long period of association with Tulane University, during which time he was part-time instructor in the Department of Chemistry, 1920 to 1921. He was instructor in Physiology in 1926 to 1927; instructor in Bacteriology and Pathology, 1927 to 1936; and Assistant Professor of Clinical Medicine, 1936 to 1942. In the latter year, he was made Professor of Clinical Medicine, which appointment he still holds. In the many years during which he has served as teacher and professor in Tulane University, he has contributed greatly by example and inspiration to the teaching and direction of medical students, many of whom are members of our Society, and those of neighboring States.

Within the field of practice Dr. Lawson has had long and careful training in the field of pathology, and has become an eminent authority. His training began as an extern in Pathology at Touro Infirmary in 1923 and 1924. The following year he was a graduate assistant in Pathology there. In 1925 and 1926 he served a general interneship at Touro, and since 1926 has been Director of Pathology at the Baptist Hospital in New Orleans. He was Director of Pathology in the Sara Mayo Hospital, 1940 to 1950. He is consultant in Pathology at the U. S. Marine Hospital since 1940.

Organized medicine owes a debt of gratitude to Dr. Lawson for his many years of service. He was Treasurer of the Orleans Parish Medical Society, 1937 to 1941, and President of the Society 1941 to 1942. He was the first President of the Louisiana Association of Pathologists in 1940-1941. He was Treasurer of the New Orleans Graduate Medical Assembly, 1946-1948, and President-Elect of the New Orleans Graduate Medical Assembly at the present time. He is on the Executive Committee of the American Society of Clinical Pathologists. He is Councillor of the State of Louisiana to the Southern Medical Association. He is a member of many units of organized medicine, including the Orleans Parish Medical Society, the Louisiana State Medical Society, the American Medical Association, the Louisiana Association of Pathologists, the Southern Medical Association, the American Society of Clinical Pathologists, the American Association of Pathologists and Bacteriologists, the American Biological Photographic Association, and the American Chemical Society. He is a founding member of the College of American Pathologists, and a diplomate of the American Board of Pathology since 1936.

Dr. Lawson is a veteran of two wars, having been a second lieutenant in Infantry in 1918, in World War I, and a lieutenant commander in the U. S. Navy in World War II.

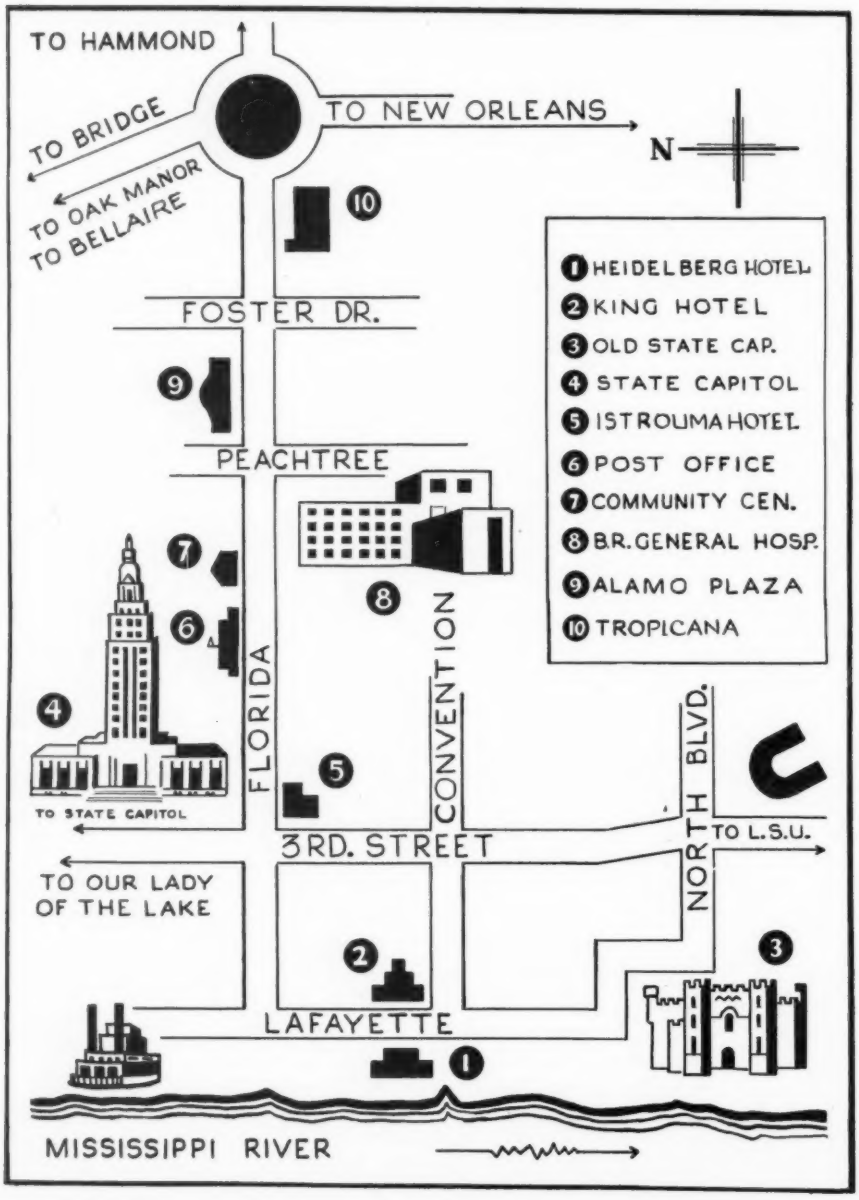
In spite of these time consuming activities, Dr. Lawson had had opportunity for social interests, which include membership in the Sigma Alpha Epsilon fraternity, the Phi Chi medical fraternity, the Army and Navy Club, and the New Orleans Country Club.

The Society appreciates Dr. Lawson's conscientious efforts in behalf of the general welfare. All are happy to remember that even though he leaves the office of President he will be available for conference and consultation to assist in many phases of the Society's efforts to improve medicine as a whole, and to combat the efforts of Communists to establish state medicine. The best wishes of the Society go with Dr. Lawson.

SCHEMATIC DIRECTIONAL GUIDE OF BATON ROUGE

ANNUAL MEETING

Complete program for the 1950 meeting of the State Society, to be held in Baton Rouge, April 24-26, will be sent to each member of the Society two weeks prior to the meeting. This will include all data concerning opening session on Monday evening, scientific sessions on Tuesday and Wednesday and meetings of the House of Delegates. A large attendance is expected at this meeting and it is felt that a review of the program will make every member interested in making plans to attend.



LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

INVITATION TO VISIT HAWAII

The Hawaii Territorial Medical Association and the Honolulu Medical Society invite visitors to the AMA convention in San Francisco to take a post-convention trip to Hawaii. They report to have arranged excellent facilities for an extension of the benefits and pleasures of the San Francisco convention and also recreational and entertainment features. The Committee on Arrangements report that the tour to Hawaii could be regarded as part of the convention under the plan suggested by the Honolulu County Medical Society to set up one evening or a half day for a clinical discussion or symposium in Honolulu on a medical subject of vital and wide interest, thus, making Hawaii visit an integral part of the San Francisco convention.

The Committee further reports that the Hawaii trip, moreover, would serve as a splendid vacation to cap the convention for the delegates and their families and suggests that a "commuter's trip" be taken due to Hawaii's proximity to the west coast—only nine hours by luxurious airliner on several daily flights, and for those with a little more time, four and one-half days by cruise ship.

Although Hawaii is famous as a year round playground, it is the summer that is truly the splendid season. There are swimming and surfing, deep sea fishing, golfing on any of 14 excellent courses on all the islands, unique sightseeing trips, or, just loafing.

DELEGATIONS FROM OUR TWO MEDICAL SCHOOLS GUESTS OF ELI LILY AND COMPANY

On February 19, 20, 21, delegations from the L. S. U. Medical School and Tulane University School of Medicine, were guests of Eli Lilly and Company in Indianapolis. During their visit, they made a thorough inspection of the Laboratories including the Research Laboratories, replica of the original laboratories, and the Biological Laboratories at Greenfield, Indiana, where demonstrations were made showing the production of biological products.

CONFERENCE ON FUNDAMENTAL CANCER RESEARCH

Meetings of interest to scientists and physicians will be held at the Texas Medical Center, Houston, Texas, May 12-13. Featured speakers will be Dr. A. C. Broders of the Mayo Clinic, Dr. Marshall Burcer of the Oak Ridge Institute of Nuclear Studies and Dr. C. P. Rhoads of the Memorial Hospital, New York City. The meetings include:

(1) the fourth annual SYMPOSIUM ON FUNDAMENTAL CANCER RESEARCH, of The University of Texas, M. D. Anderson Hospital for Cancer Research, during which one-half day will be devoted to selected papers on the subject of isotopes in cancer research;

(2) a CANCER PATHOLOGY CONFERENCE of The University of Texas Postgraduate School of Medicine, on "Tumors of Muscle Origin";

(3) a SOUTH CENTRAL REGIONAL MEETING OF THE COLLEGE OF AMERICAN PATHOLOGISTS, with co-participation in the symposium and pathology conference.

Dr. C. P. Rhoads will be the banquet speaker on the evening of May 12. Further information may be obtained from William O. Russell, M. D., 2310 Baldwin Street, Houston, Texas.

COMING MEDICAL MEETING

The International Post-Graduate Medical Assembly of Southwest Texas will hold their annual meeting in San Antonio, Texas, at the Municipal Auditorium, January 23, 24, 25, 1951. Dr. Merton M. Minter, President; Dr. John J. Hinchey, Treasurer.

POST-CLINICAL TOUR OF THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

A group of about 30 American physicians, many of whom were accompanied by their wives, making up a total of about 50 persons, arrived in Kingston, Jamaica, March 18, on a specially-chartered Pan American Airways clipper, for a four-day visit, during which they inspected hospitals and clinics and observed public health methods. They left Miami on March 11.

The stop at Jamaica was part of a two-weeks'

program which included brief calls at Puerto Rico, the Virgin Islands, and the Dominican Republic. The last stop before returning to the U. S. A. was a three-day visit in Havana.

Their aerial tour was a post-clinical feature of the New Orleans Graduate Medical Assembly, which is a gathering of practicing physicians for brief refresher courses, sponsored by about 100 New Orleans doctors. Medical men from over 35 states attended the 1950 sessions.

The doctors were specially interested in the George V Hospital, largest tuberculosis institution in the West Indies, and in the various public health and hygiene developments that have been inaugurated over a term of years with the cooperation of the Rockefeller Foundation.

Among the physicians and others arriving were Dr. and Mrs. Edmund G. Vinje of Hazen, N. D., Dr. and Mrs. Carl J. Baumgartner of Bismarck, N. D., Dr. and Mrs. Carroll Lund of Williston, N. D., Dr. and Mrs. Edward K. Chunn of Houston, Tex., Dr. and Mrs. Russell F. Weyher of Detroit, Mich., Dr. and Mrs. James A. Tesson, Dr. and Mrs. Fred Irwig and Dr. and Mrs. Edward H. Thiessen, all of Kansas City, Mo., Dr. J. Hutchings White and Dr. M. K. Thompson of Muskogee, Okla., Dr. and Mrs. Carl Granberry, Dr. Curtis Tyrone, Dr. Robert A. Robinson and Mrs. Irma B. Sherwood, all of New Orleans, Dr. and Mrs. W. Terrell Simpson and Miss Betty Ann Simpson of Winter Haven, Fla., Dr. and Mrs. Edgar H. Greene of Atlanta, Ga., Dr. and Mrs. J. Franklin Gorrell of Tulsa, Okla., Dr. and Mrs. William J. Wilson of Ogden, Utah, Dr. and Mrs. James Williams of Jacksonville, Fla., and Dr. G. S. Walker and Dr. R. S. Hollingsworth of Clinton, Mo.

SOUTHEASTERN SURGICAL CONFERENCE

Dr. Lucien A. LeDoux, Past President and Trustee of the Southern Medical Association, delivered the Annual C. Jeff Miller Memorial Medical Lecture before the Southeastern Surgical Congress at its meeting in Washington, D. C. Dr. LeDoux chose as his subject: "C. Jeff Miller, His Teachings in the Management of Uterine Fibroids."

NEW ORLEANS MEDICAL AND SURGICAL JOURNAL BACK ISSUES AVAILABLE

1929—January through December
1930—January through December
1931—January through June
1934—January through December
1935—May through December
1936—January through September
1937—Few copies
1938—January through December
1939—January through December
1940—January through December
1941—January through December
1942—January through December
1943—January through December
1944—January through December
1945—January through December
1946—Few copies
1947—January through August — November through December
1948—January through February—September through December

INFORMATION CONCERNING IMMUNIZATIONS FOR OVERSEAS TRAVEL

The State Department of Health frequently receives requests for immmunizing for travel abroad, and has issued the following summary from the bulletin prepared by the office of International Health.

Information as to which immunizations are required for travel to any particular country may be obtained from the Passport Division of the Department of State which will mail a blank international certificate of inoculation and vaccination.

The local physician can do any of the immunizations except for Yellow Fever immunizations which, because of the great difficulty of maintaining a potent vaccine, are given only at the port of embarkation or at a U. S. Marine Hospital.

Smallpox: Recommended before travel to any foreign country. Vaccinate at least 14 days before leaving the United States. Observe and record the reaction on the 3rd and 9th days. The certificate of smallpox vaccination is valid for three years. If Yellow Fever inoculations are to be given too, they should be given at least 5 days before the smallpox vaccine is given.

Yellow Fever: Give at least 10 days before leaving the country. One injection is required. Certificate becomes valid on the 10th day following vaccination and remains so for 4 years. Rather than spending 10 days or more at the port of embarkation many would prefer to go to their nearest U. S. Marine Hospital for the inoculation. (See the accompanying list for these hospitals).

Cholera: Start immunizations at least 2 to 3 weeks before leaving the United States. The standard course is 2 injections at 7 to 10 day intervals. A third injection is advisable. The International Cholera Certificate is valid for 6 months from the date of last vaccination. It can be kept in force with booster injections.

Typhus: Begin inoculation 2-3 weeks before leaving the country. Standard course is 2 doses at a 7 to 10 day interval. While the certificate is valid for 1 year, a booster dose is recommended at 6 months if the danger of typhus is still present.

Typhoid and Paratyphoid: Should be taken by every person traveling abroad. Standard course is three inoculations at 7 to 10 day intervals. Annual boosters should be taken.

Diphtheria: Children under 15 who go abroad should be immunized against diphtheria. For travel in some areas of high diphtheria incident,

adults up to the age of 35 should be immunized if their Shick tests are negative.

Note: Individuals receiving the required immunizations should bring or send their certificates to the State Dept. of Health for certification, as foreign agencies usually like the seal of a governmental agency on said certificates.

Stations of the Public Health Service Administering Yellow Fever Vaccine:

New York City, U. S. Public Health Service Dispensary, 67 Hudson Street, Barclay 7-6150.

Baltimore, Md., U. S. Marine Hospital, Wyman Park Dr. and 31st St., University 3930:

Washington, D. C., U. S. Public Health Service Dispensary, Federal Security Bldg., So., 4th and D. Streets, S. W., Executive 6300.

Miami, Florida, (1) U. S. Quarantine Station, Fishers Island, (2) U. S. Public Health Service, Relief Station, 365 Federal Building, 5-1959.

New Orleans, La., U. S. Marine Hospital, 210 State Street, Uptown 8700.

Brownsville, Tex., U. S. Quarantine Station, U. S. Border Service Building, International Bridge and 14th Street.

Chicago, Ill., U. S. Marine Hospital, 4141 Clarendon Avenue, Lake View 6340.

Los Angeles, Calif., U. S. Public Health Service, Relief Station, 406 Federal Building.

San Francisco, California, U. S. Marine Hospital, 14th Avenue and Park Blvd., Bayview 0259, 0260, 0261, 0262, 0263, 0264, 0265.

Hamilton, Mont., Rocky Mountain Laboratory, South 4th Street, Call Hamilton and ask for Laboratory.

Cleveland, Ohio, U. S. Marine Hospital, Fairhill Rd. and East 124th St., Garfield 2260.

Savannah, Ga., U. S. Marine Hospital, York and Abercorn Sts., 7714, 2-9161, 2-0162.

Fort Worth, Tex., U. S. Public Health Service Hospital, Fort Worth 5-2104, 5-2105, 5-2106.

Detroit, Michigan, U. S. Marine Hospital, Windmill Pointe, Valley 29300.

Boston, Mass., U. S. Marine Hospital, 77 Warren Street, Stadium 23400.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

The annual open meeting of the Woman's Auxiliary to the Shreveport Medical Society was held February 8. Dr. W. W. Bolton, Associate Director of the Public Health Division of the American Medical Association spoke on "Recent Medical Advances". The speaker was introduced by Dr. J. H. Eddy, Vice-president of the Shreveport Medical Society.

The Auxiliary had invited members of all the woman's clubs in the city to be their guests in order that they might hear Dr. Bolton. Following his address, the Auxiliary entertained their guests at tea.

In holding this meeting, the Shreveport Auxiliary has once again made a real contribution in the field of public relations. Their Speaker's Bureau which was organized last year to bring to club women of Shreveport facts pertaining to socialized medicine, was recognized throughout the state as having been a most commendable project.

The February meeting of the Ouachita Parish Auxiliary followed a luncheon at a local restaurant. The highlight of the meeting was a review by Mrs. James Shonlau on the "Biography of Harvey Cushing".

Mrs. Ray B. Theaux was the guest speaker at the February meeting of the Lafayette Parish Auxiliary. She gave a talk and demonstration on the making of aluminum trays and coasters. She explained where and how materials would be available for those members who cared to make trays.

A committee to make plans for Doctor's Day was appointed. It consists of: Mrs. Edgar Breaux, Mrs. J. J. Burdin, Mrs. Ralph Bourgeois and Mrs. Lee Sonnier.

The Orleans Auxiliary was hostess to the wives of doctors attending the New Orleans Graduate Medical Assembly. The ladies were entertained at a fashion show and tea which was held in conjunction with the regular monthly meeting.

Newspaper editorials and a radio program have been planned for the observance of Doctor's Day. The Orleans Auxiliary has made its usual donation of $25.00 to the Commemoration Fund in honor of the doctors. This fund is used for the care of indigent widows of doctors.

April has been designated as "Membership Month". At this meeting members who have not attended meetings for some time will be brought "back into the fold". New members will be specially sponsored by members of the board to create good fellowship and welcome them into the group.

Plans for Doctor's Day, March 30, were discussed at the regular meeting of the Woman's Auxiliary to the Calcasieu Parish Medical Society.

The Jefferson Davis Parish Auxiliary had a book review by Mrs. L. E. Shirley. She reviewed the book "Mystery, Magic and Medicine" by Howard W. Hagard, Associate Professor of Applied Physiology at Yale University. The book deals with the rise of medicine from superstition to science.

Plans are being made by Jefferson Davis Auxiliary to honor their doctors at a banquet in celebration of Doctor's Day.

In Rapides Parish, the doctors will be honored at a coffee party given jointly by the Auxiliary and Baptist Hospital. Each doctor will be presented with a red carnation boutonniere.

Mrs. Daniel M. Kingsley
Chairman
Press and Publicity

Woman's Auxiliary
Louisiana State Medical Society
Annual Meeting
April 24-26, 1950—Heidelberg Hotel

Monday, April 24, 1950

Registration ..... 9:00 A. M.
Mezzanine Floor, Heidelberg Hotel

Pre-Convention Executive Board Meeting ..... 10:00 A. M.
Conference Room, Louisiana National Bank Building, Mrs. John S. Dunn, Presiding

Invocation
Reverend Philip Werlein

Appointment of Special Committees
Auditing
Finance
Resolutions

Luncheon—Baton Rouge Country Club 1:00 P. M.

General Meeting—First Session—
Baton Rouge Country Club ..... 2:00 P. M.
Invocation
Reverend Charles J. Murphy

Address of Welcome
Mrs. Wiley A. Dial

Response
Mrs. L. L. Davidge, Shreveport, Louisiana

Reading of Minutes of last General Session

Reports of Parish Presidents

Report of Woman's Auxiliary to the American Medical Association
Mrs. Nathan Polmer, New Orleans, Louisiana

Report of Woman's Auxiliary to the Southern Medical Association
Mrs. Allen C. Winters, Alexandria, Louisiana

Reports of State Committees
Archives
Bulletin
Cancer
Commemoration Fund
Doctor's Day
Historian
Hygeia
Organization
Preservation of Medical-Cultural Items
Press and Publicity
Printing
Revision of By-Laws
Rural Health
Year Book

Adjournment

Official Opening Meeting of the Louisiana State Medical Society, Public Invited ..... 8:00 P. M.
Heidelberg Hotel Roof

Tuesday, April 25, 1950

General Meeting — Second Session — Jefferson Country Club, Mrs. John S. Dunn, Presiding ..... 9:00 A. M.
The Lord's Prayer
In Memoriam
Mrs. William Kohlmann

Benediction
Rabbi Walter G. Peiser

Greetings from Honored Guests
Dr. Edwin H. Lawson, President, Louisiana State Medical Society
Dr. George Wright, President-Elect, Louisiana State Medical Society
Dr. Daniel J. Fourrier, President, East Baton Rouge Parish Medical Society
Dr. Paul T. Talbot, Secretary, Louisiana State Medical Society
Dr. U. S. Hargrove, Convention Chairman
Mrs. David B. Allman, President, Woman's Auxiliary American Medical Association
Mrs. Arthur A. Herold, President-Elect, Woman's Auxiliary American Medical Association
Mrs. Robert C. Haynes, President, Woman's Auxiliary American Medical Association

Reports of State Committees (continued)
Editor, News and Views
Finance
Legislation
Program
Public Relations

Reports of State Officers
President-Elect
Vice-Presidents
Treasurer
Recording and Corresponding Secretaries
Parliamentarian
Chairman of District Councilors
President

Reports of Special Committees
Auditing
Finance
Registration

Unfinished Business
Recommendations of Executive Board

New Business
Report of Nominating Committee
Election of Officers
Introduction and Installation of New Officers
Announcements of incoming President
Report of Resolutions Chairman

Adjournment

Luncheon—Jefferson Country Club..... 1:30 P. M.
Tea or Reception (to be announced later)
.....5:00-7:30 P. M.
President's Dance ..... 8:00 P. M.

Wednesday, April 26, 1950

Post-Convention Board Meeting and
School of Instruction ..... 9:00 A. M.
Conference Room, Louisiana National Bank Building
Mrs. DeWitt T. Milam, Presiding

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BOOK REVIEWS

Handbooks of Diseases of the Skin: By Richard L. Sutton, M. D. and Richard L. Sutton, Jr., M. D. St. Louis, C. V. Mosby, 1949. Pp. 749, Illus. Price, $12.50.

The "Handbook," a new volume in the various texts on dermatology authored by the Suttons, is more complete than the "Synopsis" and less voluminous than the fourth edition of "Introduction to Dermatology". It seems to be primarily intended as a text for medical students but will be found useful by practitioners in dermatology.

It is particularly commendable that in this volume the authors have changed the order of presentation. Diseases are classified by their etiological factors and presented in this grouping. This leaves the chapter on "Dermatitides of Unknown Cause," to include such familiar conditions as atopic dermatitis, pityriasis rosea, lichen planus, psoriasis, and lupus erythematosus, but this chapter should shrink in size in view of rapid development in all fields of medicine.

The chapters on contact dermatitis is especially well done and the section on the treatment of syphillis is as up to date as the time of publication would permit.

Illustrations are well selected, many of them new, but some carried over from earlier volumes.

This book should be well accepted.

M. T. VAN STUDDIFORD, M. D.

Pathology and Surgery of Thyroid Disease: By Joseph L. DeCourcy, M. D. and Cornelius B. DeCourcy, M. D. Springfield, Ill. Chas. C. Thomas, 1949. Pp. 476, pl. illus. Price, $10.00.

This treatise on various aspects of the thyroid gland is based on the clinical experience of the authors, and the extensive literature on thyroid disease.

The first chapter briefly surveys the authors' impressions on various aspects of hyperthyroidism and hypothyroidism, and of the morphologic changes affecting the thyroid in which no disturbance of function is encountered. Ensuing chapters deal with historical contributions made in thyroid disease; the embryology and the anatomy, histology, and cytology of the thyroid gland. Detailed surveys of the physiological contributions and pharmacological therapeutic agents are next presented.

The pathology of the thyroid gland is presented and illustrated. The authors next present their views regarding the causation of signs and symptoms in exophthalmic goiter, (diffuse toxic goiter). They feel the "most likely explanation for the pathogenesis of exopthalmic goiter is that the condition is dependent on a disturbance of the sympathetic nervous system, influenced by various sympathomimetic agents, such as infection, psychic upset, and some obscure thyrogenic factor." Their explanation of the causation of exophthalmos, tachycardia, and the rise in BMR, is quite at variance with other authorities on these topics.

Other chapters discuss thyroid disease in the aged, preoperative considerations, anesthesia in thyroid surgery, and mention the initial contributions being made with radioactive iodine.

In some sections the book is needlessly repetitious. Too often unsubstantiated impressions of the authors are presented as facts without discussion of contrary opinions. For example, "The only truly benign tumor of the thyroid is fetal adenoma . . ." and "As the tumor grows older it tends to undergo degeneration with production of toxicity." The same criticism applies to sections on the pathogeneses of exophthalmos and exophthalmic goiter. The chapter dealing with the gross anatomy of the thyroid gland would be considerably more valuable if the text were accompanied by gross anatomic illustrations.

This book is felt to be too extensive and detailed for the medical student or general practitioner, but is recommended as a useful reference for those with specialized interest in the thyroid gland.

ROBERT RAINEY, M. D.

Campbell's Operative Orthopedics: By J. S. Speed, M. D., and Hugh Smith, M. D., Second Edition, Vol. I, St. Louis, The C. V. Mosby Co., 1949, with 1141 illustrations, including 2 color plates, 836 pages, pages 1-43: Authors' Index & Subject Index, also Table of Contents.

Campbell's Operative Orthopedics represents a monumental task of reviewing the world literature in orthopedic surgery and gleaning the pertinent information as to new operative technics and incorporating them into the information contained in the first edition of this classic in orthopedic surgery. The second edition not only includes a complete revision of the first edition but it also departs somewhat from the style of the first edition in that the authors have secured collaboration from a number of authorities in compiling this second edition. The entire staff of the Campbell Clinic has cooperated in this revision, together with such outstanding authorities as M. N. Smith-Petersen on mold arthroplasty, D. B. Slocum on amputations, and Francis Murphy on peripheral nerve surgery. The illustrations in the second edition are excellent; photographs of x-rays are considerably more creative than they were in the first edition, and the line drawings are of excellent quality.

This book represents the best in operative orthopedic procedures and should be the principal source book in any orthopedic or general surgeon's library insofar as bone joint surgery is concerned. The inclusion of the authors' opinion as to the efficacy of the various procedures is a welcomed addition in this present two-volume second edition.

JACK WICKSTROM, M. D.

Shearer's Manual of Human Dissection: Edited by Charles E. Tobin, Ph.D. 2nd. ed. Philadelphia, The Blakiston Company, 1949. Pp. 286, illus. Price, $4.50.

This manual is a dissecting guide designed to be used with additional reading in any of the standard descriptive textbooks of human anatomy. While it contains 79 line drawings a good atlas would be a necessary adjunct to its use. Dr. Tobin has succeeded in producing a manual which is neither a mere set of directions for step by step procedure in dissecting or one of the classical type with voluminous descriptions and excessive text material.

The arrangement of this book by regions allows considerable flexibility in its use. The general order of dissection can be determined to suit the requirements of the individual instructor.

The method of dissection used is very good. Structures are well exposed for study. The process is not destructive so that normal relationships may be observed, in most cases, after the procedure has been completed.

The use of bold face type throughout the text in indicating important structures and an adequate index are two features worth mentioning.

Dr. Tobin's revision of the original manual has resulted in a very much improved book.

ADRIAN F. REED, PH.D., M. D.

An Atlas of the Blood and Bone Marrows By R. Philip Custer, M. D. Philadelphia, W. B. Saunders Co., 1949. Pp. iii & 321; illustrations 285, 42 in color. Price, $15.00.

The purpose of this book, as stated in the introduction, is to set forth the principal disorders of the blood and blood forming organs, and to correlate the changes in this system with those which appear elsewhere in the body. It is thus designed for use not only by clinical hematologists, but also by pathologists, clinicians and technicians as well.

The contents are organized into several sections, treating first the mechanism of normal hematopoiesis and its relationship to the blood and bone marrow. This is followed by consideration of various disorders of the blood and bone marrow: deficiency anemias, aplastic and hypoplastic anemias, ectopia of bone marrow, hypersplenism, hemolytic anemias, ill-defined anemias and hemorrhagic states. In similar manner, there then follows consideration of effects of chemical and physical agents, leukocytosis, leukemoid reactions and leukopenia, infections, leukemia and polycythemia. The body of the book is completed by a section on hematologic technique.

One of the most welcome sections of the book is

a four-page discussion of hematologic terminology, with recommendations for usage of terms based on the findings of a committee sponsored by the American Society of Clinical Pathologists and the American Medical Association. Many of the proposed terms are not now in common usage and might well offer a source for misunderstanding, but they are individually defined in the text and should provide a definite, clean-cut point of departure for future usage. As the author points out, a multiplicity of terms should be avoided in an effort to attain consistency. This section alone, is without illustrations. While the cell forms decribed are depicted elsewhere in the volume, it might be advantageous for the non-expert reader if at least a few, somewhat idealized drawings were to accompany the descriptions.

As the name suggests, a prominent feature of this volume is its illustrations. The figures are selected from typical miscroscopic preparations of peripheral blood, marrow imprints and marrow sections. In this manner, the importance of the marrow is emphasized, as well as its relationship to the peripheral blood picture. All but a few of the figures are photomicrographs which add an authenticity not possible from use of drawings alone. Photographic illustration is quite adequqate *per se* for use by the expert hematologist, but for the less specialized observer, the photomicrograph is often deficient for portrayal of finer cytologic detail so necessary for accurate identification in many of the cell types. One method of obviating this difficulty might be the use of occasional side-by-side illustrations of a single field, where one portion of the figure depicts the finer details somewhat idealized by a drawing, and the other an actual photomicrographic reproduction of equal magnification. The bas relief figures are an excellent medium for illustration of changes seen in cells of the erythrocytic series.

Most of the photomicrographs represent considerable areas of the preparations. This is a highly desirable feature, especially in study of tissue sections where the character of various portions of the stroma is sometimes of paramount importance in diagnosis. The reproduction of large areas is less necessary in illustration of smears and imprints where it serves only to provide a larger field for observation.

Copious use is made of high power magnification (2000-2280 diameters) in these illustrations, some of which cover an entire page of the atlas. While the technique of photomicrography and reproduction is of excellent quality, the net result often leaves something to be desired since the outlines of many of the structures shown are markedly hazy. This seems to be the result of an attempt to extend a medium of illustration beyond the practical limits of its application. Many of the larger figures, especially those reproduced in color, can be most profitably studied from a distance of several feet, where the haziness largely disappears.

In both illustration and text material, this volume makes excellent use of bone marrow findings and their relation to diagnosis of hematologic disorders. Heretofore, major importance has been centered on findings in the peripheral blood alone, but here the author has been able to demonstrate the interdependence of the two elements in the art of diagnosis. Particularly pertinent in this connection is the rather detailed consideration of marrow sections, a factor which has formerly received too little consideration.

The final section of the volume deals with hematologic technique, but is confined to the methods of obtaining, preparing and examination of the bone marrow alone. In the opening paragraph of this section, the author postulates that consideration of peripheral blood technique has been adequately dealt with elsewhere. While this is true, the complete omission of the subject from the section somewhat nullifies its effectiveness for use by the non-expert. It might have been more advantageous for the reader if at least a brief summary of blood technique were considered side by side with that of the marrow, with concomitant recommendations for use of both types of material.

From a physical viewpoint, the publisher is to be strongly commended on his success in bringing out this volume. The type face is easy to read, and use of the double-column format is advantageous for covering the material with a minimum of ocular effort. The paper is of excellent quality and while it is ideal for reproduction of illustrations, one might wish for a less heavily glazed surface for the print because of the reflection factor involved.

Ralph N. Baillif, Ph.D.

PUBLICATIONS RECEIVED

W. B. Saunders Company, Philadelphia: Current Therapy 1950, edited by Howard F. Conn, M. D.; The Cytologic Diagnosis of Cancer, by the Staff of the Vincent Memorial Laboratory of the Vincent Memorial Hospital.

Lea & Febiger, Philadelphia: A Primer of Venous Pressure, by George E. Burch, M. D.

Charles C. Thomas, Publisher, Springfield, Ill.: Thrombosis in Arteriosclerosis of the Lower Extremities, by Edward A. Edwards, M. D., F. A. C. S.; Occupational Therapy, by William Rush Dunton, Jr., M. D., and Sidney Licht, M. D.

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